THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—15TH YEAR. SYDNEY, SATURDAY, SEPTEMBER 1, 1928. No. 9.

Authors of articles submitted for publication are requested to read the following instructions and to comply with them.

All articles must be typed with double or treble spacing. Carbon copies should not be sent. Abbreviations should be avoided, especially those of a technical character at times employed in ward notes. Words and sentences should not be underlined or typed in capitals. The selection of the correct type is undertaken by the Editors. When illustrations are required, good photographic prints on glossy gaslight papers should be submitted. Each print should be enclosed in a sheet of paper. On this sheet of paper the number of the figure and the legend to appear below the print should be typed or legibly written. On no account should any mark be made on the back of the photographic print. If no good print is available, negatives may be submitted. Line drawings, graphs, charts and the like should be drawn on thick, white paper in India ink by a person accustomed to draw for reproduction. The drawings should be large and boldly executed and all figures, lettering and symbols should be of sufficient strength and size to remain clear after reduction. Skiagrams can be reproduced satisfactorily only if good prints or negatives are available. The reproduction of all illustrations but especially of skiagrams entails the sacrifice of time and energy and is expensive. Authors are expected to take a corresponding amount of trouble on the preparation of their illustrations, whether skiagrams, photographs, wash drawings or line drawings. The references to articles and books quoted must be accurate and should be compiled according to the following scheme. The order should correspond to the order of appearance in the article. The initials and surnames of the authors, the full title of the article or book, the full (unabbreviated) title of the journal in which the article appears, the date of the issue (day, month and year) and the number of the first page should be given in this sequence.

Table of Contents

Symposium on Poliomyelitis.

Based on a series of lectures and demonstrations at a special course organized by the Melbourne Permanent Committee for Post-Graduate Work on March 13 and 14, 1928.

THE EPIDEMIOLOGY OF POLIOMYELITIS.

By JOHN DALE, O.B.E., M.D., B.Sc.,
Medical Officer of Health, Melbourne City.

IN the recent edition of his textbook on hygiene, Rosenau devotes a short section to the increase of infections of the central nervous system and voices therein the apprehension with which public health officers regard that increase. Poliomyelitis, *encephalitis lethargica*, polio-encephalitis, disseminated sclerosis and cerebro-spinal meningitis are the diseases with which he is concerned and as regards the two first named at least there can, unfortunately, be no doubt of their greater prevalence in recent years. According to the authorities they are not new diseases, but their activity has lately been such as to elevate them from the obscurity of "other diseases of the central nervous system" to the notoriety of notification and to be subjects of special chapters in textbooks and of separate sections in annual reports. It seems that the epidemic characters recently exhibited by these diseases are unprecedented and that these characters are in a state of flux. The apprehension of the health officer is therefore fully justified. The natural history of the viruses concerned is so ill understood that he has little to guide him in the efforts he should make to combat them.

Historical.

Various writers cite descriptions of cases which were apparently poliomyelitis occurring as long ago as 1836 and 1841 in Europe and America respectively. The official report of the New York epidemic of 1916 gives a list of outbreaks which occurred in the United States, in various European countries, especially Sweden and Norway, and in Australia during the latter half of the nineteenth century, the most extensive being the Vermont outbreak of 132 cases in 1894. The list includes a number of "outbreaks" of only four and five cases, but it is hard to believe that there were not other and larger epidemics during some of the years in question. It is very possible that there were and that medicine was too preoccupied or otherwise engaged to record them. In any case we know of no extensive epidemics occurring before the present century and it must be accepted that of late years some change resulting in the occurrence of such epidemics has taken place in the relation or balance existing between the parasitic virus and its human host. It was in Scandinavia that the first great prevalences occurred in the years of 1905 and 1906 and those countries have since continued to suffer very severely. Soon after that severe outbreaks were reported in various parts of the world. It is suggested by Rosenau that the present pandemic started in Sweden in 1881 and gradually gathering force, spread thence over the rest of the world. It seems certain, however, that the disease was already worldwide in its distribution and it is possible that the unknown influences which first operated in Scandinavia, soon became effective in other countries also.

The Virus.

The infecting organism is a filter-passing virus which Levaditi[1] in a most interesting and suggestive manner classes with the viruses of *encephalitis lethargica*, polio-encephalitis, herpes, chicken pox, small pox and rabies, as a group of neurotropic ectodermoses. Like those of small pox and rabies it is relatively resistant to drying, to glycerine and to the phenol group of antiseptics. When introduced in very various ways into certain monkeys it is capable of producing an illness with paralysis, often fatal, very similar to that occurring in human beings. It can be transmitted from monkey to monkey and when so passed its virulence rises to a certain level and remains fixed in a manner suggestive of the transition in rabbits of the *virus des rues* of hydrophobia to the exalted *virus fixe* of the Pasteur Institute. As with the other viruses, an animal that has recovered, is thereafter immune and Aycock and Kagan[2] have recently succeeded by repeated intradermal injections of virus-holding material in immunizing monkeys without producing any recognizable illness. No success, however, has followed attempts at immunization with killed virus. It is said to be a feature which distinguishes the filter-passing viruses from the larger pathogenic bacteria, that the former when killed have no immunizing properties. It is accepted that in human patients the virus can be demonstrated by inoculation of monkeys in the central nervous system and in the saliva, naso-pharyngeal washings and fæces. It is said also to have been shown to be present in the spleen, lymphatic glands and blood. Walshe[3] mentions that in the early stages of invasion of the human being lesions are found in the liver, spleen and lymphatic structures. Most writers state that the virus has been demonstrated (according to Aycock with difficulty) in the saliva and naso-pharyngeal washing of contacts, a most significant observation.

Occurrence of the Disease.

The disease affects mainly children. Rosenau states that 65% of all patients are under five years of age and only 5% over ten, but occasionally the proportion of patients over ten is much higher than this. Thus in Norway in 1911 the proportion of adult patients is given as 25% and of seventy-six patients attacked in Vermont between 1921 and 1926, as quoted by Aycock[4] to illustrate the manner of occurrence of the disease, no less than 63% were over ten years of age. Of the persons whose infection has so far been notified in Victoria during the existing prevalence 30% are over ten. Males are distinctly more susceptible to attack than females in the proportion of five to four.

Hygienic and social conditions have no effect on the incidence. It is a disease of cold and temperate rather than of warm or tropical climates, but it nevertheless occurs mainly in the warm weather, summer and autumn, though some outbreaks have occurred in the cold winter season. A most interesting feature, one more or less common to the whole group of infections of the central nervous system, is the peculiar localization. It is more frequent in scattered country populations than in crowded communities. Even in the great New York epidemic of over eight thousand cases in 1916, the incidence was highest in the less densely populated boroughs. It exhibits also very distinctly what may be described as a creeping tendency. During an outbreak the area or areas of maximum incidence slowly shift, as was seen in the weekly spot maps of the cases occurring in Brooklyn during the New York epidemic and in the extension to areas outside of the city. One could, so to speak, watch the infection spread. Similarly when a whole country is affected during successive years, the areas which suffer relatively severely in one year, are likely to escape in the succeeding years and one may infer that the susceptible material is used up, that the soil is exhausted. The most striking epidemiological feature of the disease, however, is the lack of obvious connexion between those affected. It is a rule, proved by striking exceptions, that the patients, even when becoming infected in a relatively small area, generally show no traceable connexion one with another. From the administrative point of view, that of the public health official who with his powers of quarantine, isolation and disinfection is expected to do something, "to take vigorous action to stamp it out," this behaviour is quite uncanny. Rather must he tread warily and lightly. Connected cases do occur in which infection has apparently spread directly from person to person and that the patients are potentially or actually infectious at some stage, possibly only a very early stage, must be regarded as proven, witness the rare but well substantiated milk-borne outbreaks in which a sufferer has been engaged in milking. Witness also the demonstration of virus in the nasopharynx of sufferers. But most of the cases occurring in contacts which number from 1% to 10% of the total in the various epidemics described in the literature, are now regarded by Aycock as infections from a common source. Moreover, it is very rare for doctors, nurses and attendants to become ill with the disease or for cross infection to occur in wards. In discussing the question as to whether or not the precautions usual in infective disease are or should be applied to patients suffering from poliomyelitis, James Collier[5] says: "At no hospital at which I have served nor at any London hospital at which I have been able to inquire, ... have any such precautions been taken and for the reason that experience has never called for them" and "the argument for case-to-case infection from the developed disease needs only the one indisputable case to prove it and that case is not yet forthcoming."

Whilst the absence of traceable connexion between patients is so striking a feature of the disease, it must not be overlooked that groups of connected cases do occur. These groups are of several types. One is illustrated by the outbreaks at Trästena in Sweden and at Stokes River in Devonshire, both instances in which small and relatively isolated rural communities of 500 and of 119 persons were attacked and suffered within a few weeks a high proportion of casualties totalling 10% and 30% of the respective populations. Such outbreaks postulate a high proportion of susceptible persons and their limitation to isolated communities affords evidence that most persons in non-isolated communities have acquired immunity to the disease.

Another type of grouping is illustrated by Aycock, in which a number of cases up to nine in number occur almost simultaneously in a district. Antecedent cases in the same locality may or may not have occurred to suggest a common source for the group, but incontestably such a source was there. Yet the mode of infection is a mystery.

A third type consists of the few outbreaks in which the infection was conveyed by milk. In the largest of these which took place in October, 1926, at Broadstairs in Kent, a town of 15,477 inhabitants, sixty-two cases were reported in fifteen days and practically all of the patients had consumed milk derived from one particular farm. Apparently no recognizable case had occurred on the farm in question and no suggestion is made by Aycock[6] as to how the milk became infected. In another milk-borne outbreak of ten cases at Cortland, New York, in 1925, a sufferer had actually been engaged in milking until forced to desist owing to paralysis. Undoubtedly milk may spread the disease and one is tempted to assume that the virus actually multiplies therein. Incidentally one may remark that the virus of cow pox must frequently enter milk and those of chicken pox and small pox not infrequently and yet apparently without results.

Incubation Period.

The incubation period of poliomyelitis is generally held to be variable. Formerly it was believed to be sometimes as short as three days, mainly on the evidence of multiple cases in houses and in institutions, but as mentioned above, the majority of such cases is now regarded as the result of infection from a common source; Aycock, arguing mainly from the occurrence of true secondary cases in families and institutions, now gives the period as varying from eight to sixteen days. In this connexion Aycock gives a highly suggestive footnote to his description of the Broadstairs outbreak. He was able to show that the milk supply was almost certainly infectious on October 2 to 4. The day on which the first seven patients became affected with the illness resulting in paralysis, was October 10. Several of these patients, however, suffered from transient illness about October 6 and Aycock, in the footnote referred to, suggests that this first illness was the result of systemic invasion by the

virus and corresponded to the first hump of the well-known dromedary or saddle-back temperature curve which is sometimes observed in cases of the disease. He considers that the date of onset should be taken as that of the beginning of the second period of sickness, in which the central nervous system is invaded and remarks that the first hump, the sickness of systemic invasion, is usually absent. In view of the behaviour of the disease one is inclined to go further and to point out that both phases may be so insignificant as to be overlooked and that if the first occurs without the second, as may well be the case, it would pass as an influenza or a pyrexia of unknown origin.

The Natural History of the Disease.

I may now consider the various hypotheses or alternative speculations which explain more or less the behaviour of this disease and of the other infections of the central nervous system. However dangerous it may be, a working hypothesis, an attempted integration, is inevitable. It must be assumed, in the absence of diligently sought evidence to the contrary, that there is no reservoir of infection among the lower animals, that infection is not usually carried by any insect vector and that the causative organisms are obligatory human parasites or semi-parasites. The cases of illness are merely the apparent or visible incidents in the interaction of the virus and the human species, which is continuous and constitutes the natural history of the disease, but they afford the only indication of its presence, just as the occasional leap of the porpoise above the water is a sign of the activity of the school beneath. The disease cerebro-spinal meningitis shows epidemiological characters very similar to those of poliomyelitis and, since its causative organism is relatively easily identified and has been freely studied, it may serve as a useful comparison. It is known that various closely related strains of the meningococcus are widespread among the community, that in an epidemic one strain only is likely to be entirely or mainly concerned and that carriers are very numerous as compared to sufferers from the disease, which may be expected, however, when the carrier rate rises to a certain level.

The fact that the seasonal prevalence of poliomyelitis coincides with warm weather indicates, as far as it goes, that poliomyelitis is not one of the respiratory infections, since these prevail, very properly, in the colder seasons and suggests that it is an intestinal infection incurred by ingestion. With this idea would conform also the rare instances of spread by milk, the occasional prominence of intestinal symptoms at the onset and the known infectivity of fæces; whilst the aid of the housefly in which the virus has been shown to survive for forty-eight hours, could well be invoked to bridge the gaps that exist between patients and in our knowledge. More than one mode of infection is quite possible. Were it not, however, for this late summer prevalence of poliomyelitis, it would seem that all the infections of the central nervous system, including meningococcal meningitis and the whole group of neurotropic ectodermoses, should be grouped as respiratory infections, spreading usually by transfer of oro-nasal secretion and that the differences in behaviour depend on differences in natural resistance, in the facility with which specific immunity is developed, and perhaps in the vitality or vigour of the organism, whether, for example, it flourishes or merely vegetates in a carrier.

Natural or inborn resistance is a vague conception, which covers probably a number of distinct phenomena. Against such diseases as small pox and measles there appears to be practically none and immunity developes only in response to an invasion which causes sickness, even though the virulence of the organism as evidenced by the mild nature of the illness may be very low, as in vaccinia and alastrim. But Dudley's hypothesis of the subinfective dose, which fits the facts of other diseases such as diphtheria and scarlet fever, predicates that most people possess a non-specific natural resistance which holds up a mild attack or invasion by the germs of these diseases and allows the mechanism of specific acquired resistance to come into action. Unfortunately the problem of resistance is very obscure. What is the nature of natural resistance? Does it vary from person to person and in the same person from time to time? If so, under what circumstances? Is it perchance, in regard to any germs, a function of nasal epithelium or naso-pharyngeal secretion? And does the well-known influence of diet affect natural or acquired resistance or both?

The behaviour of these infections of the central nervous system can be explained only by supposing that most persons possess natural resistance, that specific immunity readily developes and that the carrier state is frequent. According to Aycock the blood of a considerable proportion of normal persons who have not suffered from poliomyelitis, has been shown to possess specific viricidal properties, in other words to neutralize the virus of that disease. This calls to mind the high proportion of those who do not react to the Schick test among persons who have not suffered from diphtheria and suggests that the individuals have undergone a process of natural immunization. One must suppose, therefore, that the viruses are continually being carried and handed or rather sneezed, sprayed and slobbered round amongst us and that by virtue of our natural resistance and specific response most of us are made safe. Cases occur in unfortunates in whom these powers are deficient or, if normal, are overwhelmed by massive infection, probably from a florid carrier, very rarely it seems from a person in the early stage of the disease. Such a hypothesis would explain the occurrence of sporadic cases.

Epidemic prevalence requires, however, some further suppositions regarding the virus or the mechanism of resistance. It is hardly conceivable that climatic or other influences, acting directly or indirectly, could adversely affect the mechanisms of resistance against one particular organism only of the multitude which beset us. Far more likely

ILLUSTRATIONS TO DR. REGINALD WEBSTER'S DEMONSTRATION.

FIGURE I.

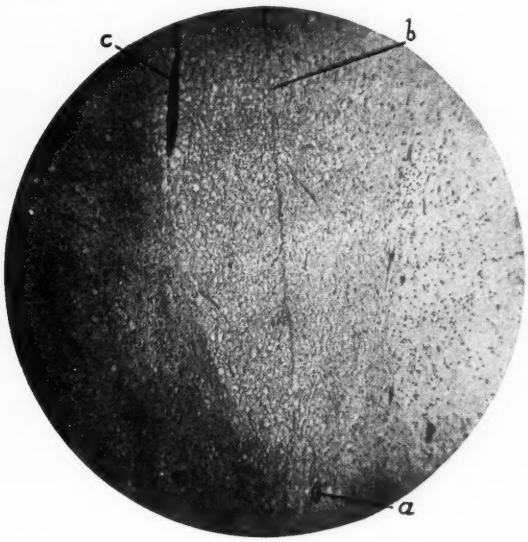

FIGURE II.

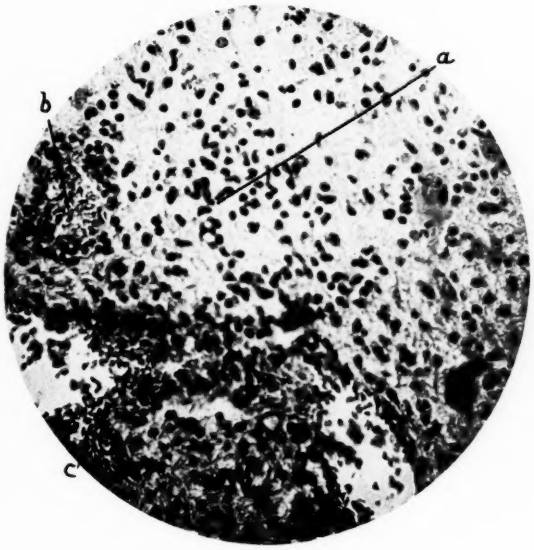

FIGURE III.

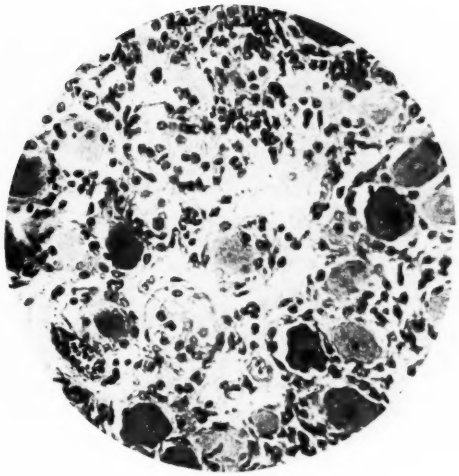

FIGURE IV.

FIGURE I: Macroscopic view of section of lumbar cord, obtained from a child *post mortem*. The specimen shows the anterior cornua delineated by hæmorrhage and illustrates an extreme rather than a common lesion. It was photographed as removed from the body without the aid of staining or any artifice to heighten the effect of the pathological process in the anterior horns.

FIGURE II: Experimental poliomyelitis. Low power photomicrograph of the spinal cord of a monkey, illustrating the manner in which the posterior horn of grey matter may be involved: (*a*) Central canal, (*b*) postero-median fissure, (*c*) perivascular cell bank in connexion with vessel entering the posterior horn.

FIGURE III: Experimental poliomyelitis. High power photomicrograph of a section of the spinal cord of a monkey: (*a*) Interstitial aggregation of leucocytes, chiefly small round lymphocytic cells, with a sprinkling of polymorphonuclear forms, (*b*) engorged blood vessel and perivascular cell collection, (*c*) interstitial hæmorrhage.

FIGURE IV: Experimental poliomyelitis. High power photomicrograph of a section of the posterior root ganglion. A few intact ganglion cells, staining well and with persisting nuclei are seen. Others stain poorly and the nucleus is just discernible. In the next degree of affection the ganglion cells are but ghosts and are undergoing neurophagocytosis. Immediately to the right of the centre of the section there appears to have been complete necrosis.

FIGURE V: Experimental poliomyelitis. Low power photomicrograph showing the path of the virus *viâ* the anterior spinal vessel in the antero-median fissure: (*a*) Antero-median fissure.

FIGURE VI: Experimental poliomyelitis. Low power field of section of the medulla of a monkey: (*a*) Choked blood vessel, (*b*) perivascular cell collarette; rarefaction of surrounding tissue presumably due to œdema.

FIGURE VII: Experimental poliomyelitis. High power photomicrograph of the spinal cord of a monkey showing inflammatory affection of meninges. The arrow heads mark the periphery of the cord and above this level the infiltration of the pia-arachnoid with small round cells is demonstrated.

FIGURE VIII: Experimental poliomyelitis. High power photomicrograph showing inflammatory lesions in the white matter of the cord: (*a*) Choked blood vessel, (*b*) focal interstitial cell collection, (*c*) axis cylinders and myelin sheaths of nerve fibres.

FIGURE IX: Experimental poliomyelitis. Low power photomicrograph of a field from the anterior cornu of the spinal cord of a monkey. Numerous ganglion cells are seen, but the majority, while preserving the general outline of a ganglion cell, appear as though they had been "dotted in." This effect is due to the fact that the cells are undergoing neurophagocytosis, as is well demonstrated in Figure X.

FIGURE X: High power representation of a portion of the field of Figure IX: (*a*) Intact ganglion cell, (*b*) ganglion cell with no discernible nucleus, (*c*) damaged cell occupied by neurophages.

ILLUSTRATIONS TO DR. REGINALD WEBSTER'S DEMONSTRATION.

FIGURE V.

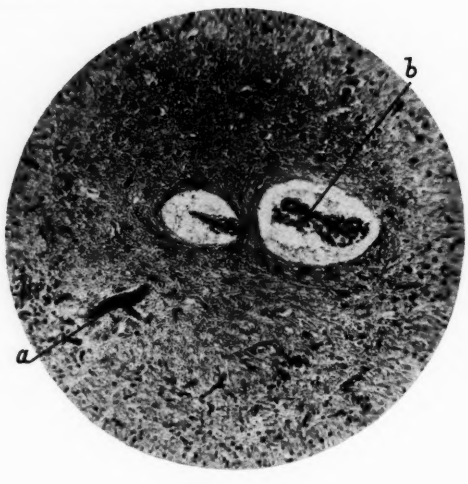

FIGURE VI.

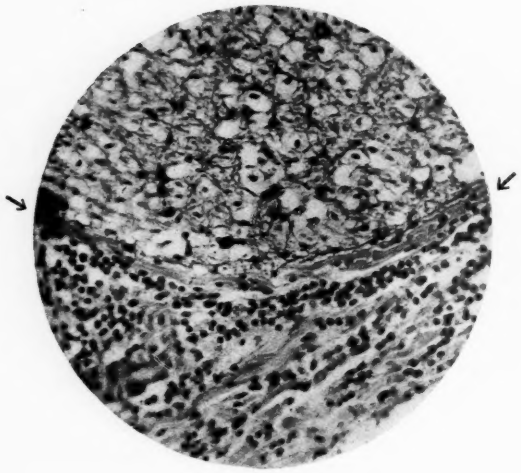

FIGURE VII.

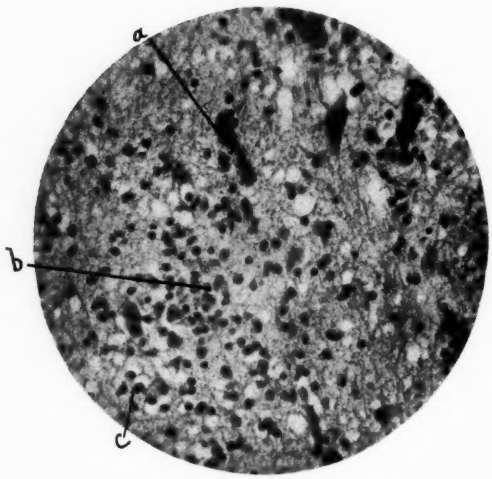

FIGURE VIII.

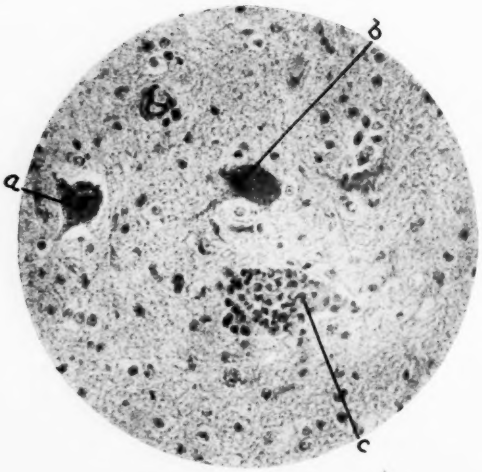

FIGURE IX.

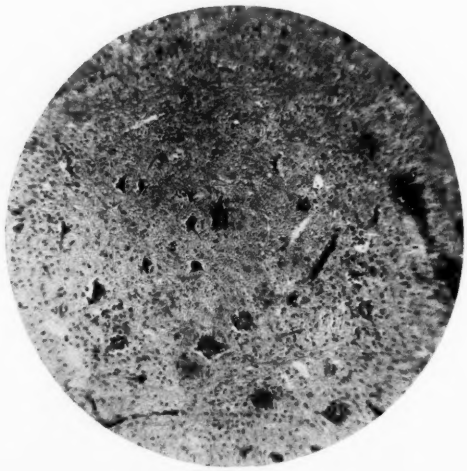

FIGURE X.

that an organism or virus undergoes some mutation, some slight shift of character or acquires some new property which upsets the balance between parasite and host. Many organisms exhibit strains that are biologically distinguishable. They must have evolved from a common progenitor and that evolution must be continuous. Some such mutation may give an organism an advantage in meeting resistance whether natural or acquired. Almost certainly the ectodermal viruses in particular are varying, especially in the degree to which they can attack the buried ectoderm of the nervous system, and this suggests another possible explanation of the increase in infections of the central nervous system. In view of Levaditi's suggestive discussion and of the recognized relation between herpes and varicella and of the recent ominous accounts of the association in many places of encephalitis and vaccinia, it is not at all far-fetched to think that the occurrence of epidemic poliomyelitis is due to the development of a more definite neurotropic tendency by a strain of an already widespread virus. The march of that particular strain, unnoticed before, would then become evident as a prevalence of poliomyelitis. As Aycock suggests, certain features of the disease, namely that young children are chiefly susceptible and that the age incidence in the country differs from that in cities in that relatively more adolescents and adults are attacked in the country, point strongly to the conclusion that, as with diphtheria, a process of natural immunization is continually going on owing to the presence of true carriers and perhaps of missed or abortive cases. This natural immunization by exposure to the subinfective dose would obviously be more complete in the crowded communities and would tend in the country to miss more children who would become susceptible adults and even to miss small communities such as those of Trästena and Stokes River.

It is significant that even in the earliest epidemics the above mentioned peculiar features were evident, so that one may conclude that when they occurred, the world population was already well immunized by strains in which the neurotropic tendency was not so highly developed. If this be so, let us hope that these strains are still amongst us and going strong!

The recent occurrence of epidemics of what were formerly rare and sporadic infections may be explained, therefore, by a hypothesis of mutation on the part of the organism. This mutation may have been effective in two possible ways in that either it gave the organism an advantage in overcoming resistance and thus actually increased the number of cases or, as seems more likely, that it was of the nature of an increase of neurotropic activity which, without increasing the number of infections, has made them more apparent because of the occurrence of conspicuous symptoms.

Other Infections of the Central Nervous System.

There are difficulties in including diseases such as *encephalitis lethargica*, Australian *X* disease and disseminated sclerosis within this nebulous hypothesis of widespread respiratory virus infection. The maximun age incidence is not among young children but among adolescents or adults. Why should young children escape as apparently they do? But there is strong evidence that the diseases are due to infection with a virus and no evidence as to the mode of entrance, so that the upper respiratory path cannot be excluded. Moreover, in the case of *encephalitis lethargica* there is no doubt of its great increase since the year 1917 and of its epidemic occurrence. Sheffield suffered over three hundred cases in one year, with no apparent connexion between the cases and with the development of the Parkinsonian syndrome in some fifty patients whose illness had not been recognized in the acute stage.[7] Truly an alarming instance of the stealth of a virus. Moreover, in contrast to the experience of Sheffield and to the usual lack of apparent connexion between cases, several instances have occurred of outbreaks in semi-isolated institutions in which a considerable proportion of the inmates were affected, suggesting a comparison with the Trästena and Stokes River outbreaks of poliomyelitis. Finally rare instances of case-to-case infection have been described. It seems highly probable, therefore, that the natural history of these diseases when it is finally elucidated, will be found to be similar and in the meantime we are forced to the unpleasant conclusion that many different viruses, with a barely concealed affection for our nervous systems, are rampant amongst us.

Measures of Control.

Regarding the control of these infections of the central nervous system it is clear that if, as is the case with meningococcal meningitis, a high carrier rate precedes and accompanies the occurrence of the disease, little is to be gained by the general application of any strict measures of isolation either of patients or of contacts. In view of the usual lack of connexion between patients suffering from virus infection and of experience of their treatment in hospital, the same holds true of them. Nevertheless, instances of apparent direct infection, both from patients and from immediate contacts of poliomyelitis have been described and it is certainly reasonable that precautions should be taken. Sufferers should, therefore, especially in the early stages be handled in such a manner as to minimize the danger of the spread of infection from them. Child contacts should not mix with other children for at least a fortnight and adults should be warned of the possibility that they may carry infection and urged to avoid as far as possible close contact with others and to be careful with regard to respiratory discharges and excreta. In country districts, especially if isolated, the possible value of such precautions is much more definite. The temporary closure of schools and a maximum interruption of social intercourse and avoidance of personal contact may well be justified.

Since milk can carry the infection, special care should be taken to prevent the consumption of raw

milk which might have become contaminated. It is, however, obvious that the prospects of any really effective control of these infections by the methods of isolation are very limited and the experiences of diphtheria and scarlet fever suggest that a solution may have to be found in artificial immunization. According to Netter[8] this has been successfully attempted in Sweden by David, who has since 1924 injected seventy-three contacts with three cubic centimetres of human immune serum. Among those thus treated only one case of transient sickness occurred, whilst of eighty-four control contacts, similarly exposed and of similar age groups, fourteen developed the disease. This experience indicates that if ample stocks of serum were available, it might now be possible to prevent a proportion of the contact cases, which as stated, constitute up to 10% of the total in an epidemic. The idea is well worth considering, especially when the disease appears in isolated country districts.

References.

[1] C. Levaditi and S. Nicolau: "*Ectodermoses Neurotropes. Études sur la Vaccine*," *Annales de l'Institut Pasteur*, 1923, Volume XXXVII, page 1.

[2] W. L. Aycock and J. R. Kagan: "Experimental Immunization in Poliomyelitis," *Journal of Immunology*, August, 1927, page 85.

[3] F. M. R. Walshe: "Poliomyelitis," *The Lancet*, February 12, 1927, page 326.

[4] W. L. Aycock: "The Epidemiology of Poliomyelitis; with Reference to Its Mode and Spread," *Journal of the American Medical Association*, 1926, Volume LXXXVII, page 75.

[5] James Collier: "The Epidemiology of Poliomyelitis," *The British Medical Journal*, September 10, 1927, page 468.

[6] W. L. Aycock: "Milk-borne Epidemic of Poliomyelitis," *American Journal of Hygiene*, November, 1927, page 791.

[7] E. W. Goodall: "A Text-Book of Infectious Diseases," 1928, page 334.

[8] Netter: *Bulletin de l'Académie de Médicine*, 1928, Volume XCIX, page 151.

PATHOLOGICAL ASPECTS OF POLIOMYELITIS.

DR. REGINALD WEBSTER gave a lecture-demonstration on the pathological aspects of poliomyelitis at the Children's Hospital. He said that a discussion of the pathology of poliomyelitis should embrace consideration of the virus, the mode of infection, pathogenesis, morbid anatomical changes and clinical pathology.

In regard to the virus very strong claims had been put forward by the Rockefeller School for the globoid bodies as the infecting agent in poliomyelitis. The evidence in favour of such rested on the demonstration in a glycerinized emulsion of the spinal cord of a virus which was capable of inducing experimental poliomyelitis in monkeys. The clinical and anatomical changes in the experimental disease very closely paralleled those in the human subject. The virus was classified among the filter-passers in view of the fact that the filtrate obtained by passing the cord emulsion through a Berkefeld filter was also capable of infecting monkeys. Morphologically the globoid bodies were extremely minute coccoid forms of size at the limit of visibility even with very powerful lenses and microscopes. They had been successfully cultivated by the Noguchi anaerobic technique and subcultures of the ninth generation had been potent in inducing experimental poliomyelitis. The globoid bodies had also been demonstrated directly in sections of nervous tissue.

It had been argued by E. C. Rosenow that the globoid bodies were merely aberrant forms assumed by a certain streptococcus when subjected to the conditions of growth prescribed for the cultivation of the globoid bodies. An antipoliomyelitic serum obtained from horses immunized against this particular streptococcus was used with apparent success by Nuzum and Willy during an epidemic of poliomyelitis in 1917. Herzog claimed to have induced the clinical picture and pathological lesions of poliomyelitis by the inoculation of animals with Rosenow's streptococcus. Many workers of high repute, however, had been unable to support the streptococcus as the ultimate ætiological factor in poliomyelitis and the weight of experimental evidence was without doubt in favour of the Rockefeller School.

The prevailing view of the mode of infection in poliomyelitis was that the ingress of the virus was effected *viâ* the nasal mucosa. Poliomyelitis had been communicated experimentally to monkeys by means of irrigations of the naso-pharynx of subjects of the disease and of healthy contacts, the concentration of the washings *in vacuo* and intracerebral inoculation of the residue. With a virus adapted to the monkey mere painting of the nasal mucosa was sufficient to induce the disease. It had been abundantly proved in the investigation of the extensive epidemic in New York in 1916 that infection was spread by healthy carriers and subjects of the abortive type of the disease. One specific observation related to the demonstration of the virus in the naso-pharyngeal secretions of both the parents of a child stricken with poliomyelitis.

Dr. Webster said that he thought that he could best convey an idea of the pathogenesis of poliomyelitis by drawing an analogy between that disease and cerebro-spinal meningitis. He emphasized that poliomyelitis was a systemic infection in the course of which paralysis might or might not occur. Cerebro-spinal meningitis furnished a parallel in that in the meningococcal infection the disease might run its course to recovery or death without any localization of the microorganisms in the meninges ever having been effected. The same held for poliomyelitis. In experimental poliomyelitis induced by the intravenous injection of the virus it had been determined by sacrificing animals at varying periods after the injection that the virus was withdrawn from the blood first by the spleen and bone marrow, secondly by the posterior root ganglia and finally by the central nervous tissue itself.

In the spontaneous disease in human subjects the virus, having reached the upper respiratory mucous membrane, multiplied locally until a point was reached at which it invaded the blood stream. As

long as the meningo-chorioidal defensive mechanism remained intact, the virus could not reach the subarachnoid space, but this line of defence could not resist indefinitely accumulation of the invading microorganisms in the blood stream. Once the virus reached the subarachnoid space, it circulated in the cerebro-spinal fluid and was conveyed along the perivascular sheaths of the vessels of the brain and cord into the substance of the cerebro-spinal axis.

At this point Dr. Webster explained the meningo-chorioidal protective function as demonstrated by Flexner and Amoss.[1] Working with a poliomyelitic virus of which five hundred cubic centimetres were necessary to produce lesions in a monkey when given intravenously, these observers had found that if the injection into a vein were combined with lumbar puncture and the subthecal injection of normal horse serum, an intravenous injection of one-tenth the amount (fifty cubic centimetres) of virus sufficed to induce poliomyelitis in a monkey of the same weight. Further, the intrathecal injection of normal monkey serum, saline solution and even such physiologically balanced fluids as Ringer's and Locke's solutions, operated in a similar manner to promote localization in the central nervous system of poliomyelitic virus introduced into the blood stream. The removal of cerebro-spinal fluid from one monkey and its replacement by that of another monkey sometimes but not invariably promoted infection, but simple lumbar puncture in the absence of trauma as evidenced by blood in the fluid, was found not to precipitate the infection of the central nervous system.

It was an everyday experience that some degree of trauma was occasioned by lumbar puncture. If hæmorrhage into the subarachnoid space should result, the conditions became the same as those in the experiment in which horse serum was introduced into the theca, blood in that situation being foreign protein.

In the case of the intrathecal injection of a foreign protein, such as horse serum, a consequent simple, irritative, aseptic meningitis could be readily understood, and the experiments of Flexner and Amoss showed that this mild disturbance of the meninges was sufficient to disorganize the meningo-chorioidal defensive mechanism to such a degree that infection of the central nervous tissues by intravenous injection of the virus was greatly facilitated. But that what would appear to be the infinitesimal pathological changes following the injection of Ringer's solution and normal monkey cerebro-spinal fluid, should similarly undermine the meningo-chorioidal protective complex, served to emphasize the extremely delicate nature of this system and its capacity for very fine adjustment.

The experimental findings had an important bearing on therapeutic intrathecal injection of serum. It followed that unless the serum introduced were specifically an immune serum, it might operate in a manner quite the reverse of beneficial by promoting the localization of the virus in the central nervous system.

After mentioning the hyperplasia of the spleen and lymphatic glands, lymphoid tissue of the small intestine and the occurrence of general cloudy swelling as the expression of systemic infection, Dr. Webster proceeded to demonstrate by means of a series of lantern slides (see figures) the morbid histology in the central nervous system. He intimated that he had prepared the microphotographs from monkeys in which he had studied the experimental disease at the time of the small epidemic in Melbourne in 1918. The perivascular cell collarette, interstitial inflammatory foci and all degrees of damage in the ganglionic cells of the anterior cornua were illustrated. An interesting slide related to the lesions observed in the posterior root ganglia; the changes in these tissues were in all respects comparable with those in the grey matter of the cord. It was demonstrated that the changes were by no means confined to the anterior horns of the grey matter and that the inflammatory reaction was to be seen in the posterior cornua, the white matter and the meninges.

The clinical pathology of poliomyelitis resolved itself into the laboratory investigation of the cerebro-spinal fluid. It was perhaps unnecessary to point out that the fluid as a rule remained clear, that a pleocytosis was noted, that an increase in protein content occurred and that a veil-like coagulum separated at times on standing. The formation of the coagulum was hindered by agitation of the fluid and by cold storage.

The number and character of the cells present varied with the stage of the disease. The highest cell counts were to be observed when lumbar puncture was performed about twelve to eighteen hours after the invasion of the nervous system. The clinical signs of such invasion consisted of severe headache, meningism, the spine sign of Draper or paralysis. Cell counts at this time might total as many as 2,500 per cubic millimetre and in general it had been found that a high count seriously affected the prognosis. When the lumbar puncture was not performed until some days had elapsed after the invasion of the central nervous system, the count of cells in the cerebro-spinal fluid lost its prognostic value, although it might still be of use in diagnosis.

An increase in the number of cells in the cerebro-spinal fluid occurred regularly before the onset of paralysis. In such preparalytic stage 50% and upwards of the cells present were polymorphonuclear cells. This represented an important point of distinction between poliomyelitis and *encephalitis lethargica* in which condition the pleocytosis, always moderate and rarely exceeding one hundred cells per cubic millimetre, was purely mononuclear.

After the onset of paralysis, although the cell count remained high for a few days, the lymphocytes usually outnumbered the polymorphonuclear leucocytes which tended to disappear rapidly after paralysis was established.

In poliomyelitis as a rule there was no diminution in the percentages of glucose and chlorides in the cerebro-spinal fluid.

Dr. Webster said that at the Children's Hospital his experience had been that the diagnosis of poliomyelitis usually turned on the elimination of tuberculous meningitis. For this purpose he was accustomed to search for tubercle bacilli and estimate the total chlorides in the cerebro-spinal fluid. Normally the chloride content, expressed as sodium chloride, lay between 720 and 750 milligrammes per hundred cubic centimetres. A progressive diminution of the total chlorides was a very reliable index of tuberculous meningitis. With a chloride content of 650 milligrammes per hundred cubic centimetres or less he would consider a diagnosis of poliomyelitis untenable as against tuberculous meningitis.

Dr. Webster next proceeded to demonstrate the technique of cell counting with the aid of large sized drawings illustrating the rulings in the Fuchs-Rosenthal counting chamber and the Buerker hæmocytometer chamber. Whatever counting chamber was used, it was necessary to count the cells in at least one cubic millimetre of cerebro-spinal fluid. The identification of the character of the cells was simplified by staining the nuclei with a stain such as toluidine blue or thionine blue. One part of stain was added to nine parts of cerebro-spinal fluid and the mixture allowed to stand for a few minutes prior to counting. The proportions of cerebro-spinal fluid and stain indicated were very conveniently adjusted in the pipette designed for diluting blood in order to make a leucocyte count.

The Fuchs-Rosenthal counting chamber was ruled as a large square of double lines, sixteen square millimetres in area. This was divided again by double lines into sixteen smaller squares, each of which was one square millimetre in area, and each of these again was divided by single lines into sixteen smaller squares. The depth of the cell was 0·2 millimetre and its cubic content therefore $^{16}/_{5}$ cubic millimetres. With a dilution of fluid of 9 in 10 the quantity of cerebro-spinal fluid lying over the squares would be $^{16}/_{5} \times {}^{9}/_{10}$ or $^{144}/_{50}$, approximately three cubic millimetres. The cell count per cubic millimetre, therefore, was obtained by dividing the total number of cells in the chamber by three.

In the absence of the Fuchs-Rosenthal counting chamber, especially designed for the enumeration of cells in cerebro-spinal fluid, accurate counts could be made with the Buerker hæmocytometer. By covering both the ruled areas in this apparatus the number of cells in eighteen separate squares, each one square millimetre in area, could be counted. As the chamber was 0·1 millimetre deep an estimate of the cells in 1·8 cubic millimetres of the stained fluid was thereby obtained. With a dilution of nine parts of cerebro-spinal fluid to one of stain the quantity of cerebro-spinal fluid in which the cells had been computed would be $1{\cdot}8 \times 0{\cdot}9$ cubic millimetres. The cell count per cubic millimetre was obtained by the formula $\frac{X}{1{\cdot}8 \times 0{\cdot}9}$.

The qualitative detection of excess protein, with globulin as the index was as a rule all that was necessary. Globulin was most conveniently demonstrated by the Ross-Jones modification of the Nonne-Apelt reaction (Phase 1).

In conclusion Dr. Webster detailed the technique for the estimation of the total chlorides. He emphasized the necessity for a dry test tube for the reception of the fluid if a chloride estimation were desired. If a small quantity of fluid, such as five cubic centimetres, were collected in a wet test tube, a 10% dilution of the fluid might be effected. The 10% reduction in chlorides thus brought about would be very misleading.

The principle underlying the method for the estimation of the total chlorides, which was very simple, was the precipitation of the chlorides as insoluble silver chloride. As soon as all the chloride was thus precipitated, red silver chromate was formed between the silver nitrate used for titration and the indicator, potassium chromate. The silver nitrate was prepared by dissolving 5·814 grammes of pure silver nitrate in a litre of distilled water (or more conveniently 2·5 grammes silver nitrate in 430 cubic centimetres of distilled water).

In carrying out the estimation two cubic centimetres of cerebro-spinal fluid were pipetted into fifteen or twenty cubic centimetres of distilled water and a drop or two of 10% potassium chromate added. The silver nitrate solution was run in from a burette, the flask being kept agitated. The end point of the reaction was indicated by a permanent change of colour from lemon to orange yellow. Each cubic centimetre of silver nitrate used indicated one gramme of chloride per litre of cerebro-spinal fluid or one hundred milligrammes per hundred cubic centimetres. Thus, if the titration figure were 7·2 cubic centimetres, the chloride content would be 720 milligrammes per hundred cubic centimetres, which was the usual method of expression.

The members of the class then adjourned to the laboratory where facilities were provided for cell counting and chloride estimations. In the laboratory demonstration Dr. Webster was assisted by Dr. B. A. Hunt and Dr. J. Fone.

Reference.

(1) S. Flexner and H. L. Amoss: *The Journal of Experimental Medicine*, 1917, page 525.

THE PREPARATION OF SERUM FROM HUMAN DONORS RECOVERED FROM POLIOMYELITIS.

By F. G. MORGAN, M.B., B.S. (Melbourne),
Director of the Commonwealth Serum Laboratories, Melbourne.

MY talk this afternoon will deal briefly with the preparation of curative serum from human donors for the treatment of poliomyelitis. It may be of interest to you if I preface the technical details with a few remarks upon the immunity developed in experimental animals following inoculation with the living virus. The virus may be obtained by

making a 5% suspension of brain or spinal cord of a patient who has succumbed to the disease. The mixture is then filtered, a suitable candle being used. The disease can be reproduced in a susceptible animal, such as a monkey, by intracerebral, intrathecal, intraperitoneal and in a small percentage of cases by subcutaneous injection of the filtrate.

Flexner and Noguchi succeeded after many failures in cultivating under anaerobic conditions a minute globoid organism which they claimed was the cause of poliomyelitis. Their cultures appeared to lose virulence after seven or eight transfers in artificial media.

Rosenow and others isolated a pleomorphic streptococcus which at one stage resembled the globoid bodies of Noguchi and passed through Berkefeld candles. Bull, however, failed to reproduce the disease with these cultures. They are now generally regarded as secondary invaders, since they have been recovered from the nervous tissue of persons succumbing to diseases other than poliomyelitis.

One attack of the disease confers immunity in both man and the monkey. In those rare instances in which two distinct attacks have been reported in the same person, there has been a questionable diagnosis as to the nature of one or other of the attacks. Early in the study of the disease it was found that the blood serum from persons after recovery had a virucidal property. The serum from experimentally inoculated monkeys which have recovered, protected other monkeys, either mixed *in vitro* with the virus before injection or given separately to the animals before or shortly after the injection of the virus.

Aycock and Kagan claimed that it was possible to protect monkeys by the intracutaneous injection of active virus. Twenty-one out of twenty-three animals so treated were found to be resistant to subsequent intracerebral inoculation. It has not yet been found practicable to immunize animals successfully in order to obtain hyperimmune serum which would confer a passive immunity upon affected children.

The use of convalescent serum of both human beings and monkeys has been shown of value in the treatment of the disease whether naturally acquired or artificially produced.

Monkeys are small animals and the quantity of blood which can be obtained from them, is insufficient for practical therapeutic purposes. As far as present knowledge goes, the use of convalescent human serum is the only practical means of aborting the disease in affected children. For the best results the serum should be given in the preparalytic stage.

It is necessary to point out here that the amount of blood available from persons recovered from poliomyelitis is strictly limited. It is impossible to obtain sufficient serum locally to enable us to act in Melbourne as a distributing centre for such a preparation for the whole of Australia or even for Victoria.

Donors are hard to obtain. The quantity of blood one can remove from an individual patient is strictly limited and only a small number of successive bleedings can be made from the same patient.

The serum is somewhat expensive to prepare, chiefly owing to the source from which it is obtained. The yield of serum is small—only about 150 cubic centimetres being available as filtered serum from each bleeding.

I shall now describe the stages in the preparation of the serum. The exhibits on the table show the various stages in treatment of the blood after it is received at the laboratories.

The patient is bled at the hospital and a special collecting apparatus is used. The technique of removal and the apparatus have already been fully described in the "Transactions of the Australasian Medical Congress (British Medical Association)," Dunedin, 1927. The article in question appears on page 263 of the Transactions which appeared as a supplement to THE MEDICAL JOURNAL OF AUSTRALIA, with the number dated October 8, 1927.

The blood flowing from the vein is collected in the vessel containing oxalate solution which is gently agitated during the whole bleeding to insure that the anticoagulant is thoroughly mixed with the blood to prevent clotting.

When the whole blood is received at the laboratories the specimen is kept free from disturbing influences, so that the red cells may sediment to the bottom. When a good separation of red cells from plasma has occurred, the supernatant plasma is syphoned off. A sufficient quantity of calcium chloride is added to the plasma and a clot quickly forms which, when complete, is gently separated from the side of the vessel with a sterile glass rod or platinum loop. Contraction of the clot is assisted by placing a brass weight on the top of the clot to press it flat.

These stages can be seen in the exhibits here. The first tube contains the sedimented red cells with supernatant plasma. To the second tube which is separated plasma, I shall now add the required quantity of calcium chloride. You will observe the rapid formation of the clot. We shall allow it to stand until the end of the demonstration, when you will see the separation of the clot and further shrinking, leading to a complete separation of the serum. In the third tube can be seen a brass weight pressing on the fibrin clot, which is pressed down at the bottom of the tube.

Strict aseptic precautions are observed to maintain sterility throughout the process from the time of bleeding the patient until the separated serum is passed through a sterile filter candle. The apparatus used is sterilized in steam under pressure or in a hot air sterilizer, according to the nature of the material used in its composition.

The specimens of serum from separate donors are arranged in groups and those of the serum of the same group are pooled; the pooled sample is filtered as serum of one group through Pasteur Chamberland F. candles into collecting bottles.

Serum is classified as belonging to one of four groups, according to the length of time which has elapsed since the donor has suffered from the disease.

Sterility tests for the presence of bacteria are made upon the bulk bottle and later when the serum is bottled into ten and fifteen cubic centimetre hermetically sealed ampoules, further tests for sterility are performed. These tests are designed to detect the presence of both anaerobic and aerobic organisms both at 37° C. and at 20° C.

To guard against the transfer of infectious disease from the donor to the recipient, the medical practitioner who bleeds the patient, examines the donor to insure that he is free from such diseases as measles, scarlet fever, endocarditis and other infections. The blood is examined for the Wassermann reaction in every donor; further safeguards are the filtration through Pasteur Chamberland F. candles and sterility tests.

The somewhat elaborate method of preparation of the serum which we have employed, has not been adopted up to the present as far as I am aware by other workers preparing this serum. The precautions taken have given us a product the use of which has not been attended by any unpleasant symptoms or reactions in the recipient, whether given intrathecally, intravenously or subcutaneously.

Dr. Macnamara has supervised the greater part of the work of administering this serum to patients and has been in close touch with medical men using the serum, if she has not directly supervised the administration. She informs me that no reaction of any kind has been experienced.

THE SERUM THERAPY OF POLIOMYELITIS.

By JEAN MACNAMARA, M.D. (Melbourne),
Clinical Assistant to Physician to Out-Patients, Children's Hospital, Melbourne.

IN 1910 in their experimental work Flexner and Lewis[1] and Landsteiner and Levaditi[2] demonstrated that the blood of persons and monkeys who had recovered from an attack of poliomyelitis, contained immune substances capable of neutralizing an otherwise fatal dose of the virus of poliomyelitis. In 1911 Flexner and Lewis demonstrated that the intraspinal injection of human or monkey convalescent serum protected monkeys from an otherwise fatal effect of an intravenous injection of the virus. As experimental poliomyelitis induced in the monkey is a much more severe and more frequently fatal malady than the disease in man, it was reasonable to assume that even better results would be obtained from the use of immune serum, human or monkey, in the disease in man than the results obtained from its use in the laboratory animal. It was demonstrated that these antibodies appeared within twelve days of the onset and in one instance were present after an elapse of thirty-four years. Immune principles appeared in the blood of persons after abortive or mild attacks, who had been in contact with patients definitely suffering from paralysis. The original work of Netter and Levaditi has been confirmed in America and Sweden. In 1914 Netter and Levaditi described a case of poliomyelitis in the human treated with convalescent serum and by May, 1917, the number of patients treated in France had reached forty-seven. Netter used daily intrathecal injections for eight to fifteen days in small doses and he concluded that in the preparalytic stage the use of serum averted paralysis, while he considered the administration of serum was justified after paralysis had occurred in the ascending type.[3] In 1916 in New York serum was used by Draper, Peabody and Zingher and later by Amoss and Chesney.[4] [5] Better results were obtained in the series of patients treated by Amoss and Chesney when intrathecal and intravenous injections were combined and the dosage was over sixty cubic centimetres.

In New Zealand in 1924 and 1925 convalescent or human immune serum was used extensively in Wellington and Dunedin and other centres. At the close of the epidemic a *questionnaire* was issued by the Department of Health to all its district offices to gain information on various aspects of the epidemic. The replies to the questions have been published in full in the Transactions of Congress, but conclusions 17 and 18 may be quoted here.

> Conclusion 17.—Human convalescent serum was found to be a specific in producing prompt recovery and preventing paralysis if given in the preparalytic stage.
>
> Conclusion 18.—Early diagnosis by clinical observation and by lumbar puncture is essential for effective serum therapy.[6]

In Wellington, in the early stage of the epidemic, nearly all the patients were admitted to hospital after the advent of paralysis; but after a few weeks, as a result of the awakened interest of the public and a recognition by the profession of the syndrome of the preparalytic stage, 60% were admitted to hospital at a suitable stage for serum treatment. No ill effects were observed from lumbar puncture or serum administration. Under favourable conditions all the patients received intrathecally ten to fifteen cubic centimetres of convalescent serum at body heat given very slowly by gravity after the removal of from fifteen to twenty cubic centimetres of cerebro-spinal fluid, also very slowly. The older patients received in addition twenty cubic centimetres of serum intravenously and the injections were repeated within twenty-four hours if the condition required it. Dr. W. S. Robertson, who was in charge of the patients admitted to the Wellington Hospital, has written:[7] [8]

> A Pasteur serum arrived too late to be tried out. The results of our own serum were so satisfactory that one hesitated to branch out on an untried brand. As to the results of serum therapy, one cannot be dictatorial owing to the fact that there is no concrete clinical evidence that any given case is not going to be one of the abortive type without any treatment. There are dozens of cases of this type, many of whom on clinical grounds should become severely paralysed, but don't. Personally I am convinced that serum administered in the preparalytic stage, will abort an attack. One clinically typical preparalytic case was admitted when our serum stock was low. Her condition was identical with all the other cases who were apparently aborting with serum. The available doses

were given to other apparently more serious cases and that case is still with us, the most extensively paralysed one we have. Serum was given later, but, of course, too late. Every serum-treated case, treated with serum in the preparalytic stage, with one exception made a complete recovery or cleared up with a transient paresis or slight residual weakness.

While in New Zealand in February, 1927, I saw the patient to whom Dr. Robertson referred, and had the opportunity of discussing with the latter the difficulty in obtaining statistical evidence of the value of any method of therapy in a disease which, in any given case diagnosed clinically in the preparalytic stage, with confirmation of the diagnosis by the examination of the cerebro-spinal fluid, may terminate in complete recovery or in death or in paralysis of varying degree as an intermediate possibility.

After making repeated accurate counts of the cell content of the cerebro-spinal fluid, Draper was unable to note any correlation between the increase of the cells and the severity of the paralysis. In some patients affected this year with widespread paralysis, the cell count has been under fifty cells per cubic millimetre, while in others with the mildest paralysis the cell counts ranged around five hundred cells per cubic centimetre in the same phase of the acute stage. Because of this uncertainty of prognosis, we cannot disregard the clinical impressions of those who have treated many patients with serum and many without it.

If I may digress, I should like to outline the history of serum therapy for poliomyelitis in Victoria.

During the 1918 epidemic an attempt was made at the Children's Hospital to organize serum treatment. A circular letter was sent to thirty-five former patients, the subjects of poliomyelitis within the preceding five years. Seven responded, the mothers of three of whom objected to the blood-letting. As the remaining four were small children, Dr. Webster was unable to obtain blood in sufficient quantity to use except for experimental purposes.[9]

Early in 1924 a little boy was admitted to the hospital with a history of fever, irritability for two days, following vague illness during the preceding week. He had neck stiffness and Kernig's sign and the provisional diagnosis was tuberculous meningitis. Very soon after admission, however, it was noticed that he had weakness of the dorsiflexors of the left wrist and a flaccid paralysis rapidly developed. Lumbar puncture was performed; the fluid was under increased pressure, clear and contained excess of globulin and an increase of cells, eighty-six per cubic millimetre, the majority being mononuclear cells; the diagnosis was obviously poliomyelitis. Twenty-four hours elapsed before his back, arm and leg muscles became paralysed. Four days after his admission his playmate living in a house in the same street was admitted to hospital with a story and symptoms similar to those of his friend and he developed paralysis on the third day of his illness. Both these children had a definite preparalytic stage; in both the diagnosis was confirmed by the examination of the cerbro-spinal fluid; both became extensively paralysed while under observation and though splinted at once and treated since, they have extensive residual paralysis as evidence of the damage done to the anterior horn cells within those first few days. Draper's book "Acute Poliomyelitis" was obtained and after the results obtained by Amoss and Chesney in the treatment in the preparalytic stage with human immune serum had been read, it seemed rather a disgrace that we should have stood back and allowed these children to become paralysed without attempting to do more than apply splints to insure complete rest. Later in 1924, interest was brought back to the subject by the reports in the lay press of the epidemic in New Zealand. In 1925 during January and February when many cases were still occurring in New Zealand, the number of patients admitted to the Children's Hospital in Melbourne was greater than the number admitted during the autumn for several years. Cables were sent to the Director-General of Health of New Zealand, Dr. Valentine, and as by March, 1925, convalescent serum had come to be regarded as a specific in the preparalytic stage in New Zealand, through the initiative of Mr. Kent Hughes, arrangements were made for the preparation of a supply in Melbourne. The initial difficulties encountered have been described in the Transactions of Congress,[10],[11] but the experience gained in 1925 has made the difficulties of obtaining a much larger supply of serum in 1928 much less formidable. The tedious work of tracing donors was saved; the adults affected in 1925 had been kept under observation during the three years and valuable help has been given by the 3LO Broadcasting Company in tracing others who had moved. As in 1925 Dr. Morgan decided to aim at the preparation of a product, sterile and free from added antiseptic and as free as possible from hæmolysis. The freedom from reaction after the injection of large amounts of serum, intrathecally and intravenously, of our patients treated in 1925 compared so favourably with the reactions reported after the injection of serum to which tricresol had been added, that it was considered worth the extra trouble involved to insure sterility by other means. Various technical difficulties have been overcome by the use of rustless steel needles of a large calibre, or of an ordinary large steel needle sterilized in the autoclave after coating with a paraffin of low melting point, such as "Nujol," by pointing the needle towards the hand, by teaching the donor before the puncture how to contract and relax the forearm muscles rhythmically without jerking the needle from the vein, yet with sufficient force to cause a rapid flow of blood through the needle and tubing with each contraction. I am indebted to Dr. Tebbutt and to Mr. McLure for much helpful advice on points of technique, which has made it possible to remove four hundred and fifty to six hundred cubic centimetres (fifteen to twenty ounces) of blood by veni-puncture. Venesection was not considered advisable because of the

added risk of contamination and hæmolysis and because the necessity for dressing the small wound would make the process unpopular. In February difficulty was experienced in obtaining blood in sufficient quantities in Melbourne to provide sufficient serum to cover the needs of the State. As the greater incidence of the disease among adults has been in the country, our supply was augmented through the valuable cooperation of Dr. Hewitt, Dr. Langlands, Dr. D. D. Brown and Dr. Simpson by blood taken in the country from adults who could not be brought to Melbourne. The same technique and the same apparatus for collecting were used, the apparatus being prepared at the Commonwealth Serum Laboratories. In other countries, France, America and New Zealand, this laborious and expensive method we are using was not undertaken. Blood was allowed to run from the needle into a sterile vessel and to clot; the serum was allowed to separate and was pipetted or syphoned off, inactivated and used at once. To enable serum to be stored with safety for use in remote country districts, this more elaborate method was evolved. At present a small stock of serum in ampoules is stored at the following country centres in Victoria: Ballarat, Bendigo, Mildura, Wangaratta, Swan Hill, Colac, Drouin, Hamilton and Coleraine, so that the delay inseparable from the transport of serum from Melbourne may be eliminated in the event of a patient seeking treatment in the preparalytic stage. This serum has not been concentrated in any way. Its potency is low. All serum depreciates more quickly at room temperature than at ice box temperature. In view of the difficulty of obtaining large supplies, an endeavour has been made to conserve the strength of the serum by keeping it even during transport, at ice box temperature. Arrangements have been made with the Victorian Railways for renewal of the ice at various junctions as required. In *The Lancet* of February 12, 1927, it is recorded that Dr. Walshe, in opening a discussion of the Medical Society of London, referred to the use of human convalescent serum and the good results obtained from its use in Europe. He concludes that since human serum alone is available for this purpose, we have not yet a means of providing sufficient serum to cope with the demands of a severe epidemic. In Melbourne during the summer and autumn of 1928 thirteen litres of blood have been obtained from convalescents and old patients, principally the adults affected in 1925 and the serum yield has varied between 40% and 50%. Had the demand for serum been greater, larger quantities could have been obtained and after the experience gained this summer, it is anticipated that if the need for very large amounts of serum should ever arise, it could be met, if sufficient funds were available to provide for the necessarily heavy expenses and the payment of the donors. The cost of the serum at present is estimated at about two shillings per cubic centimetre.

In remote country districts of America serum is prepared locally as required from persons in the neighbourhood who have previously had poliomyelitis. This method, while simple and inexpensive, is not free from the disadvantage that several hours' delay is inseparable from the process of separating the serum and the possibility of transmitting syphilis or other blood-borne infection has to be recognized. Whereas in the case of the serum prepared at the laboratories a Wassermann test has been carried out with every specimen of blood obtained from each donor and the preparation of the serum is held up until Miss F. E. Williams, at the Walter and Eliza Hall Institute, or Dr. Rennie, at the University, has reported that no reaction has been obtained. The process of filtration through a Chamberland F. candle should eliminate the possibility of transmitting any infectious disease and after each bleeding the donors are kept under observation for some weeks. Rosenow and his collaborators have prepared a serum by immunizing horses against strains of streptococci obtained from human beings with poliomyelitis. This work lacks the experimental basis of the immune human serum and, as far as I know, Rosenow's serum has not been used in Australia. It is hoped that later a method will be perfected of immunizing an animal, such as the horse, against poliomyelitis. In 1919 in New York Neustaedter and Banzhaf[3] and in 1918 in Paris Petit injected first in the sheep and then in the horse progressively increasing doses of the cords of monkeys inoculated with the virus of poliomyelitis. The serum of these horses is said to be as effective experimentally as human convalescent serum and it has been used extensively in epidemics in France and Roumania. The results are said to be satisfactory, though the reaction following intrathecal injection is greater than that which follows injection of human immune serum. Until more details have been published the possibilities of usefulness of serum prepared from this source cannot be judged.

The Preparalytic Stage.

A discussion of serum therapy cannot disregard the diagnosis in the preparalytic stage. In times of epidemic this may be easy. For example one child in a household or street is affected with paralysis; a few days later an illness in a contact, with slight fever, irritability and drowsiness would lead one to suspect poliomyelitis. The type of case which has occurred in Victoria in 1925 to 1928 has not been the fulminant, without warning of paralysis, but rather the dromedary or straggling types described by Draper. Retention of urine, photophobia and localized sweating have been frequently observed this summer. In times of epidemic, when a patient, child or adult, in whom a very careful clinical examination fails to reveal any other cause for the illness, such as pyelitis or pneumonia, after a variable degree and duration of illness developes more irritability and the sign described in America as the spine sign, a lumbar puncture is justified. The method of eliciting the spine sign is important. If one delays until the degree of neck stiffness and of Kernig's sign which accompaniees meningitis is

present, the diagnosis will often be missed in the pre-paralytic stage. The spine sign is a voluntary disinclination to flex the spine anteriorly because any anterior flexion of the spine causes pain. When older children are asked to touch the knees with the lips they will try and give up the attempt because of the pain induced. With a little child the surest way to elicit this sign is to take him on one's knee, reassure him by very gentle handling and then encourage him to attempt to obtain some desirable toy held in such a position that anterior flexion of the spine is necessary to obtain it. Each voluntary effort is checked if the spine sign is present and the child gives up the attempt. Adults affected within the last three years have described the intense back-ache and pain in the back of the neck which precedes paralysis. In examining a child suspected to be suffering from poliomyelitis, time spent in reassuring him and making friends with him and an appreciation of the fact that his limbs are tender and any disturbance of position is unwelcome, will not be wasted. These children are quick to discriminate between a person who handles them gently, and another adult who makes an examination hastily, just as a child threatened with respiratory paralysis is quick to appreciate the difference between a nurse who spares him any exertion and another who has to be asked for attention. The characteristic attitude described by Amoss has been observed frequently this summer.[12] If there is no involvement of the respiratory centre and no other contraindication, the patient is allowed to sit on a chair or on the side of the bed. This attitude is characteristically assumed. The back is held straight and both hands rest upon the bed slightly behind the buttocks with the arms stiff and straight in the attempt to take the weight from the painful back. When the patient is asked to bend over and place the head between the knees, he bends only from the hips.

In performing lumbar puncture, which is justified if this sign has developed, one should remove only the minimum quantity of fluid required for the examination and be prepared to make that examination forthwith. As demonstrated by Dr. Reginald Webster in 1920[13] in pneumococcal septicæmia in the laboratory animal, the removal of large quantities of cerebro-spinal fluid may promote infection of the meninges by impairing the integrity of the chorioid plexus. In any septicæmic condition the removal of large quantities of fluid, unless this is followed very shortly by the administration of immune serum, is to be deprecated, for the alteration of the meningo-chorioidal defence by the sudden withdrawal of fluid tends to precipitate an invasion of the cord or meninges. Three cases illustrating this point have occurred this year. In these lumbar puncture was performed and a large amount of fluid withdrawn in the preparalytic stage, in which a diagnosis was made clinically and an interval of twelve hours, eighteen hours and fourteen hours respectively allowed to elapse before the administration of serum. These cases will be published in detail later. Paralysis developed in each child. They are referred to in this paper to emphasize the importance of the avoidance of delay in examination of the fluid.

To overcome this difficulty we have followed in Melbourne the procedure adopted in New York in 1916,[14] of taking to each patient seen in consultation outside of the public hospitals the apparatus, for immediate bedside examination of the fluid, cell count, cell staining and globulin test and a sterile kit for lumbar puncture. If lumbar puncture be performed under local anæsthesia, care should be taken to avoid injecting the "Novocain" along the track of the puncture needle into the spinal canal, for a slight admixture of anæsthetic and fluid will render the count inaccurate. If the clinical findings justify a lumbar puncture, then the microscope and slide are adjusted ready for use, the needle inserted and two cubic centimetres of fluid withdrawn; the obturator is replaced in the needle to prevent the escape of any more fluid and the examination is made at once. If the cells are not increased, the needle can then be withdrawn. If the fluid is clear or ground glass in appearance and the cells are increased and the mononuclear type predominates, unless the clinical history is suggestive of tuberculous meningitis, serum can be given at once intrathecally after the removal of a larger quantity of fluid. The serum should be warmed, given very slowly and by gravity. Thus the patient is saved a second lumbar puncture. The estimation of the chloride content can, if necessary, be carried out before a further quantity of serum is injected intravenously. As far as we can judge from reported cases and from the experience in Victoria in 1925 and 1928, the best results have been obtained by a combination of intravenous and intrathecal administrations in doses varying according to the size of the patient, but not less than fifty cubic centimetres. In California, Shaw, Theland and Fleischer gave intramuscular injections of convalescent serum in the preparalytic stage with prevention of the paralysis in seven patients.[15] During this summer when the age of the child has made intravenous injections difficult, the supplementary injections have been given intramuscularly.

In cases of the Landry type with ascending paralysis, if large quantities of serum are available, it should be given, provided that in assessing the results and considering the value of this method of therapy, the two groups of cases are clearly differentiated. In 1925 in three patients in whom paralysis was steadily advancing, no further extension occurred after the administration of serum; but this summer four rapidly progressing cases have gone on to a fatal conclusion despite the administration of serum after paralysis had occurred. Nor is this surprising in view of the microscopic changes around the vessels of the cord, the cutting off of the vascular supply to areas of the cord. Once the invasion of the cord has advanced to such a degree that paralysis is evident clinically, there is no certainty that any immune principles injected into the blood stream will reach the site of the lesion. In this type of case the method suggested by Amoss

and Aycock[16] of combining intravenous injections of hypertonic saline solution with intrathecal injection of immune serum may prove useful in reducing the œdema of the cord. In adults extension of paralysis may occur after an apparently quiescent interval of days or even weeks, the "jump" variety of Batten.[17] If a patient, particularly adult or older child, developes a secondary rise of temperature which cannot be explained by other causes, such as pyelitis or pneumonia, a second injection of serum should be given, particularly if the fever be accompanied by an increase of meningeal signs or hyperæsthesia or localized pain in a limb. In Victoria this year it was considered wiser to limit the administration of serum to patients thought to be in the preparalytic stage or limb paretic stage or to adults with the ascending type. The recognition by the profession of the syndrome of the preparalytic stage has given opportunity for its use. The results will be published in detail later.

Acknowledgements.

I would take this opportunity of acknowledging my great indebtedness to the members of the profession in Victoria, whose cooperation has made this work possible, to Dr. W. S. Robertson, of Wellington, for advice on many technical points, and to Dr. Arnold Netter, of Paris, for literature.

References.

(1) S. Flexner and P. A. Lewis: "Experimental Poliomyelitis in Monkeys," *Journal of the American Medical Association*, May 28, 1910, page 1781.
(2) Levaditi and Landsteiner: *Comptes Rendus des Séances de la Société de Biologie*, 1910, Volume LVIII, page 311.
(3) A. Netter: *Bulletin de l'Académie de Médicine*, January 31, 1928, page 147.
(4) George Draper: "Acute Poliomyelitis," 1917.
(5) H. L. Amoss and A. M. Chesney: "A Report on the Serum Treatment of Twenty-six Cases of Epidemic Poliomyelitis," *Journal of Experimental Medicine*, 1917, Volume XXV, page 581.
(6) C. M. Hector: "Poliomyelitis," "Transactions of the Australasian Medical Congress," Supplement to THE MEDICAL JOURNAL OF AUSTRALIA, October 8, 1927, page 258.
(7) W. S. Robertson: "A Review of the Cases of Polioencephalitis in the Wellington Hospital," *New Zealand Medical Journal*, October, 1925, page 235.
(8) W. S. Robertson: Letters not yet published.
(9) Reginald Webster: "Poliomyelitis: Pathological Notes and Experimental Study," THE MEDICAL JOURNAL OF AUSTRALIA, January 11, 1919, page 21.
(10) F. G. Morgan: "Collecting Blood from Donors and Preparation of Curative Serum for Use in Poliomyelitis," "Transactions of the Australasian Medical Congress," Supplement to THE MEDICAL JOURNAL OF AUSTRALIA, October 8, 1927, page 263.
(11) Jean Macnamara: "Treatment of Poliomyelitis by Means of Human Immune Serum," "Transactions of the Australasian Medical Congress," Supplement to THE MEDICAL JOURNAL OF AUSTRALIA, October 8, 1927, page 265.
(12) H. L. Amoss: "Tice System of Medicine," Volume V, page 349.
(13) Reginald Webster: "Lumbar Puncture and Meningitis," THE MEDICAL JOURNAL OF AUSTRALIA, November 27, 1920, page 487.
(14) New York Health Reports, 1916.
(15) E. B. Shaw, H. E. Thelander and E. C. Fleischner: "Convalescent Serum in Pre-paralytic Cases of Poliomyelitis," *The Journal of the American Medical Association*, November 14, 1925, page 1555.
(16) W. L. Aycock and H. L. Amoss: "Experiments on Specific Therapy in Poliomyelitis; the Utilization of Hypertonic Solutions in the Serum Treatment of Experimental Poliomyelitis," *Bulletin of the Johns Hopkins Hospital*, 1923, Volume XXXIV, page 361.
(17) F. Batten, A. Garrod and H. Thursfield: "Diseases of Children," 1913, page 768.

THE TREATMENT OF POLIOMYELITIS DURING THE ACUTE AND CONVALESCENT STAGES.

By JEAN MACNAMARA, M.D. (Melbourne),
Clinical Assistant to Physician to Out-Patients, Children's Hospital, Melbourne.

As suggested by Lovett, poliomyelitis after the development of paralysis may conveniently be divided into three stages for the purpose of treatment.

1. The acute stage, dating from the onset of the illness until the disappearance of tenderness, a period varying from a few days to three months.

2. The convalescent stage, beginning at the end of the acute stage and continuing as long as recovery of function is proceeding, usually up to the end of two years.

3. The chronic stage, after the termination of the convalescent stage, when the condition is more or less stationary.

Treatment During the Acute Stage.

Serum therapy has been discussed in detail. Drugs afford little help. "Hexamine" may be given, but its usefulness is doubtful and hæmaturia is readily induced in children by its administration, unless the drug is given in small doses, freely diluted. Instillation of 10% "Argyrol" into the nostrils has been advised as a prophylactic measure for patients in whom nasal discharge is a feature. It appears to do no harm. Sedatives such as "Dial" are frequently more useful than morphine in inducing rest in the first few days. Morphine may be needed for the severe backache and limb pains which are frequently complained of by the adult patients. Catheterization may be necessary for some days. If bulbar involvement is threatened, it is wiser to give repeatedly glucose and saline solution *per rectum* than to worry the patient with repeated attempts at swallowing. With older children and adults fluid will be taken more readily if given through a bent glass tube to obviate the anterior flexion necessitated by other vessels. The patient should be nursed on a hard bed with a firm mattress, a cradle being used to support the weight of the bed clothes. Frequently a restless child is made comfortable and enabled to sleep by means of a pillow placed under the shoulders in such a way as to induce a slight degree of opisthotonus. From the outset rest should be as complete as possible and nursing disturbance reduced to a minimum. In Wellington plaster beds were made for all patients under four years of age and for any with spinal or abdominal weakness. Plaster bed boots, extending above the knees, with the feet at right angles were made for older children, the limbs being steadied by sandbags. The arms should be placed in a position of shoulder

abduction, the elbows semiflexed, the forearms semiprone. It is usual to place the upper limb in a position of full outward rotation. If this be carried out at the onset and paralysis developes, the limb should be carefully examined and the power of the outward rotators compared with that of the inward rotators of the shoulder joint and the position modified to prevent stretching of the weaker muscle group. The plaster bed possesses the advantages of a supported relaxation of the whole of the body in the position of physiological rest: it is cheap and can be prepared quickly. The double Thomas's splint and its modifications are, however, more useful when the paralysis is severe and modifications of the limb positions are required as recovery occurs. If the splint is made with joint in the vertical stem so that the degree of abduction of the lower limbs can be easily altered, the same splint can be made to insure recumbency throughout the convalescent stage. It is well, if arm pieces are fitted, to have them made of iron soft enough to be moulded by the hands and for little children with severe paralysis the addition of a head piece adds greatly to the comfort. The type in which support is given by movable slings passed from one side to the other and attached to the steel or metal outline of the head piece, possesses the advantage that the hair is not thinned by continued friction on leather.

A brisk aperient, a good supply of fluid, a firm bed and immediate splinting are the chief points in the treatment during the acute stage. The disappearance of hyperæsthesia varies widely in different patients and as long as it persists, it is wise to insist on complete rest and to avoid massage and manipulation. After the first few days the comfort of the patient is increased by the use of warm saline baths, provided that the necessary movements are carried out without anterior flexion of the spine. Little children can easily be lifted into a bath on a sheet or board; for older children and adults the gutter bath, as used for typhoid fever patients, is very helpful in reducing the pain and tenderness and giving the patient a feeling of well-being. In giving a bath to a patient in whom the deltoids are involved, care should be taken to prevent adduction of the shoulder during immersion. For patients with this condition a modification of the gutter bath has been provided, by means of boards fencing off the sides of the bed from the level of the patient's neck to some distance beyond the feet and the large mackintosh draped over these boards as it is draped over the pillows in the ordinary gutter bath. By this means the water paddock is wide enough to allow for abduction of both shoulders during immersion in the salt water. These baths should be continued daily throughout the acute stage and well into the convalescent stage. Frequently the first evidence of returning power in a muscle is manifested in the warm fluid medium. With babies and little children in whom orthodox muscle reeducation is at first a difficult matter, the mother or nurse can be shown how to use various birds and fishes made of rubber or celluloid to encourage voluntary movements of an affected muscle. Throughout this period the splinting should prevent the development of deformities which develope so quickly and insidiously if no precautions are taken, particularly the common scoliosis adduction of the shoulder, flexion of the hip and outward rotation of the lower limb, flexion of the knee and foot drop.

Treatment of the Second Stage.

When tenderness has disappeared, the patient enters into the second stage in which a great recovery of function should be expected. The aim of treatment is twofold. The first object is to keep the muscles in the best position for return of function as a proportion of the anterior horn cells innervating the muscles recovers, with the subsidence of œdema and disappearance of the leucocytic aggregation. The second object is to educate muscle bundles by means of carefully graduated attempts at voluntary movements to undertake more work than each bundle carried out before some were put out of action. The fact that recovery occurs during the first two years and sometimes is considerable after the lapse of six months, renders the second aim feasible, for if the degree of recovery of muscle function depended on the recovery of life of the proportion of the cells not badly damaged, it would surely be completed at the end of the few weeks or at most months necessary for the subsidence of the œdema and disappearance of the leucocytic aggregation from around the vessel walls. The degree of recovery can be estimated accurately by means of Lovett's spring balance test which can be quickly and easily carried out after some practice, and which furnishes more satisfactory evidence of recovery than clinical impressions gained from observing patients at long intervals.

"The Treatment of Infantile Paralysis," by Lovett, and "Muscle Training in Infantile Paralysis," by Wright, are invaluable as guides in the treatment during the long convalescent stage. These books are almost unobtainable in Melbourne at present. I shall quote freely from the articles therein on the treatment at this stage.

In many children affected in 1925 whose treatment by rest and muscle reeducation has been continued, the return of power, as evidenced by the muscle pull of the spring balance and by increased function, has been as evident in the third six-month period as in the second. The findings of Lovett have been borne out repeatedly in other 1925 patients. "Fatigue of the affected parts of the neuro-muscular mechanism is very easily induced and is a detrimental factor of the highest importance." The custom of allowing a child to walk as soon as walking is possible, even with the help of braces, does not take into account this question of fatigue. It is obvious that walking on muscles reduced to half their normal power will easily induce fatigue. The situation was described very aptly by Charles Fayette Taylor in a book on infantile paralysis published by him in 1867. "The legs of a paralysed child in relation to its body may

be compared to those of an infant called upon to support the trunk of a man. They cannot do it and should not be asked to do it until they have become equal to their load."

A weakened muscle can be given all the use it requires by therapeutic exercises without weight-bearing and the recumbent position in such a splint as the modified double Thomas's described, reduces the risk of the development of deformity. "Patients with involvement of leg or spinal or abdominal muscles should not be allowed to walk in the first year after the attack." On general principles ambulatory activity should be undertaken only when return of power has been perfect in mild cases or when there is reason to believe that the hope of complete recovery or further substantial gain must be abandoned. The mild conditions are often disappointing; for although they offer the greatest hope of complete recovery, it is always difficult to secure the cooperation of the parents to prevent the child from walking and the early weight-bearing constitutes such gross overuse of the leg muscles that the weakness becomes permanent. It is useful to have some method of classifying the muscle power, such as: (i) Muscles incapable of any contraction, (ii) muscles capable of overcoming friction only when assisted (very poor), (iii) muscles capable of overcoming friction of joint and table surface, (iv) muscles capable of overcoming gravity alone, (v) muscles capable of overcoming gravity and outside force.

Muscle Reeducation.

In paralysis, the beneficial effects of muscular contraction on the circulation may be in part supplied by massage, radiant heat, passive movement and so forth. Wherever there is, however, the ability of a muscle to contract even slightly by an effort of the will, the muscle cells are more favourably affected by this contraction than by quickening of the circulation by any other means. If a lively circulation is started in the muscle before it contracts, the contraction will be attended by greater benefit. For this reason it is advisable in treating these patients to make use of such therapeutic measures before giving the exercises, even when voluntary contraction is good. In poliomyelitis certain nerve cells supplying a muscle being destroyed, the remainder, being unaccustomed to work together, perform their work badly and without coordination. The aim of reeducation is to train these nerve and muscle cells to work together with precision. "Parts which have functioned together tend more easily to function together again." Each time a partially paralysed muscle contracts, it improves, not only the nourishment of its fibres, but also the coordination of the nerves which stimulate it. The following are practical points in the treatment:

The patient should never be expected to carry out his exercises alone. The response of nerve and muscle is dependent on the strength of the stimulus and the volition of the patient is greatly aided by the word of command.

The paralysed limb should be uncovered, as the action of muscles cannot be seen through the clothing.

The exercises should be carried out on a hard, smooth, horizontal surface. If a table cannot be obtained, a bed may be used if large sheets of hard millboard or three-ply wood are available. When powdered these present a smooth surface with little friction.

The patient should carry out the exercises in a room alone with his teacher. The concentration required to obtain the best contraction possible cannot be expected if other people are in the room.

A muscle should be given an exercise as hard as it is capable of performing and when it has outgrown that exercise, the next in order of difficulty should be taken up.

Exercises should be carried out once daily for six days a week, if possible. It is usual to perform each movement ten to twelve times, with an interval to allow of recovery from fatigue between the contractions.

When contractions of joints exist, they should be corrected before exercises are attempted. When a weakened muscle is kept on the stretch by contracted antagonists, there is no possibility of strengthening it until the resistance has been removed.

Miss Andrew and Miss Taylor have followed this technique since 1925 and Miss Andrew and Miss Hutchinson will demonstrate in detail the methods of exercising various muscles. The position in which the child is placed, is modified according to the muscle's strength. For example if the *quadriceps femoris* be strong enough to overcome friction without assistance, but not strong enough to contract against gravity, the child should lie on the side opposite to the affected quadriceps, a pillow should be inserted between the knees and a large sheet of three-ply wood or millboard should be placed upon the pillow; the affected limb should be placed thereon and the knee bent. Until the patient is able to straighten the limb completely from the fully flexed position by means of a steady, even movement, the board should be kept horizontal. As power returns, the muscle can be given harder tasks by tilting the board gradually by the pillows under it, until the knee can be straightened against gravity. The same position is used for the hamstrings, the hip flexors and hip extensors. The person directing the exercises should discourage "I can't" and make the patient feel that "he hasn't yet, but he soon will."

The treatment of the second stage of poliomyelitis may be summed up as follows:

1. Obtain the early cooperation of the parents for long continued rest and treatment to achieve the best result of which the patient is capable.

2. When the tenderness has gone, commence muscle reeducation.

3. Radiant heat and saline baths are useful. The former can be cheaply improvised by means of the

ordinary wooden or iron cradle, some asbestos and carbon lamp.

4. Splinting must be constantly supervised and modified in order to prevent stretching of the weaker groups of muscles.

5. Deformities develope quickly and insidiously. Scoliosis, hyperextension of the great toe and adduction of the shoulder are easily corrected at their first appearance; if neglected, they may require operative interference later.

6. If abdominal weakness be present, a cloth corset or belt may be necessary, even during recumbancy, to prevent stretching of the weakened muscles which would prejudice their recovery.

7. Standing may be allowed for a few minutes daily after some months to stimulate the sense of balance, but this should not be allowed if a position of deformity is assumed in order to stand.

8. Sitting, which favours flexion of the hips, scoliosis and foot drop should be avoided.

9. The number of cases in which complete recovery is to be obtained in infantile paralysis, is greatly extended by keeping the patient from walking during the first year.

10. When it is decided that the time has come to allow the patient to walk, a brace should be worn by every patient when an unnatural position of the leg is assumed in standing or walking.

11. If the patient is of school age, arrange at the outset of this treatment for regular lessons. The Department of Postal Education of the Education Department of Victoria makes arrangements for instruction to be carried out at home in order that when treatment in bed has been completed the child will not be handicapped still further by loss of education. The adults affected usually improve to a greater extent than the children similarly affected, if given the same prolonged treatment, but with limited hospital accommodation the problem of their treatment in Victoria has yet to be solved.

THE CONSTRUCTION OF PLASTER MOULDS.

By HUGH C. TRUMBLE, M.C., M.B., B.S. (Melbourne), F.R.C.S. (England), F.C.S.A.

Honorary Surgeon to Out-Patients, Alfred Hospital, Melbourne; Honorary Surgeon to Austin Hospital, Melbourne.

THE practitioner of average ability who will take the trouble to master the simple technique required to construct plaster moulds, slabs, beds and so on, will have at his disposal a means whereby he can furnish at small cost and with little trouble splints for almost every conceivable ailment which may require the application of supporting apparatus. He will have the satisfaction of knowing, once such a splint is properly made, that it will do the work for which it was designed with a minimum of attention as it can be applied in only one, the correct position. Most of the splints figuring in textbooks of surgery which are not entirely useless or worse than useless, require the services of a skilful splint maker (a very rare specimen) in the manufacture and there after ceaseless attention on the part of the attendant to keep them in position.

It is not feasible in a short paper to give more than an outline of the methods used. As an instance and for the sake of demonstration suppose that it is required to construct an apparatus which will support the spine and the whole of one lower limb. It could easily be extended to include both lower limbs, together with one or both upper limbs.

Materials Required.

1. The amount of plaster of Paris will vary with the size of the patient. For an adult about nine kilograms (twenty pounds) will suffice. Any grade of plaster which will set will do. There is no advantage to be gained from the use of fine impression plaster such as is used in dental practice.

2. Eight ounce scrim or Hessian, to be obtained at any decorators. One metre and three-quarters (two yards) will suffice.

3. A strip of gauze large enough to overlap in all directions by six inches the parts to which the splint is to be applied.

4. Strips of aluminium cut to the desired shape, if it be intended to include such in the finished apparatus.

5. Bowls for mixing, gowns, knives, pillows and sand bags for supporting the limbs during the application.

Preliminary Preparation.

The scrim is cut into strips which correspond in area to that of the skin surface which is to be covered. For the spinal portion of the splint under consideration the strips should extend from the lower cervical area to the middle of the sacrum and from one anterior axillary line to the other across the back. For the leg extension the strips should be of about the length from sacrum to ankle joint, of width equal to one half of the circumference of the thigh. About eight of each kind should be cut out. There is no need to shape the strips, as surplus corners can be folded over where necessary.

Position of the Patient.

The position of the patient is of first importance, as once the splint is made, it cannot be altered. The patient is placed prone on a long narrow table, supported on pillows or sand bags. The distribution of these supports will vary with requirements, but usually they are placed under the forehead, upper thorax, pelvis and ankle joint. The arms must be only slightly abducted from the sides, otherwise the scapulæ will be moved and there will be trouble later. The arrangement gives a slight extension of the vertebral column with slight flexion at the hip and knee joints. If abduction is desired at the hip, the leg is carried out accordingly.

The patient being posed in the required attitude, the gauze strip is placed over him so as to overlap generously the area of skin to be covered. All is then ready for the application of the plaster. A

large dish half full of water is placed on the table or on another table quite close to the patient's pelvis. Plaster is dusted into the water and mixed thoroughly until a thin creamy consistency is reached. An assistant immerses a few of the scrim strips in the cream and hands them one at a time to his chief who lays them over the appropriate area on the patient. Plaster cream is freely poured on each layer so as to fill completely the interstices of the Hessian and leave no air spaces. Where necessary the edges of the strips are folded back. The upper edge of the splint crosses the lower cervical spine and a projection passes forwards over each shoulder. In the shoulder region the plaster should not extend far laterally or it will cause discomfort. The lateral margin should correspond more or less to a line drawn from the centre of the clavicle over the shoulder through the central point of the spine of the scapula and curving forward across the margin of the *latissimus dorsi* on to the lateral wall of the thorax. The strips passing down the leg are made to overlap the dorsal strips and to alternate with these.

The weak points in this particular apparatus are opposite the scapulæ, the hip joint and the knee joint. During the construction, therefore, extra strips are inserted in these regions, with consequent increase in thickness and strength. The strips may be folded and refolded as required to increase the thickness in any particular area. An endeavour should be made so to distribute the plaster that the finished article is strong where necessary, but not clumsily thick where no great strain will fall. Thus, in the leg extension the plaster should be at least two and a half centimetres (one inch) thick, in the mid-line of the limb posteriorly opposite the hip joint a little less at the knee joint, still less between these joints and it should fade away to about six millimetres (one-quarter of an inch) in thickness at the edges and at the ankle region. Similarly in the interscapular region the mould should be at least two and a half centimetres thick, but should become thinner towards the lateral margins.

Usually by the time the strips have been placed in position, the plaster is beginning to set. The margin of the apparatus is rapidly and easily bound at this stage by folding back and fixing with plaster the overlapping portions of the first gauze strip. Should it appear necessary to cut away any of the hardened plaster, as for instance in the region of the anus, the edge should not be bound in this region until after this has been effected. After a few minutes the mould may be lifted and removed.

With a sharp knife any desirable trimming is effected and the binding of the edge is finished. The inner or skin surface is inspected and any ridges or projections which may appear likely to cause trouble, are rubbed smooth by the flat of the operator's hand. Should it be necessary, the gauze lining is secured by smearing with a little very thin plaster cream.

In cases of urgency the mould may be used in its wet state after an hour or two. As a rule it should be placed in a warm corner and dried. To give it a fine hard surface which is impervious to water and permits of washing, the dried plaster is painted over with a thin solution of shellac in methylated spirit (about three coats).

Should it be desirable to incorporate metal strips, as for instance to provide a malleable foot piece extension, an aluminium strip of the required dimensions is placed between two of the layers of the leg extension during constructions of the same. The strip should extend to the knee region in order to acquire adequate fixation. Thereafter the projecting portion of the strip may be bent as desired.

When lying in the mould the patient should not wear any garment the removal of which will necessitate removal of the plaster. A pyjama coat should be worn back to front. All that is permissible between skin and plaster is a very thin material such as one layer of silk. Thicker cloth is liable to cause discomfort by wrinkling. Cotton wool should on no account be used.

Should there be any evidence of pressure over a bony point, a cross is made on the skin with indelible pencil. The plaster in the area is moistened with water and replaced on the patient. The pencil will make a mark on that area of the mould which corresponds to the irritated skin spot. The surface of the plaster is then scraped away over an area of about five centimetres (two inches) in diameter and to a depth of six millimetres (one-quarter of an inch).

The apparatus may, of course, be varied in an infinite number of ways to meet requirements. Once it has been constructed correctly, it will carry out efficiently the work for which it was designed. It may be removed to permit of massage, exercises and toilette of the skin and re-applied even by an unskilled person only in the correct position.

SPLINTING IN ACUTE CASES OF ANTERIOR POLIOMYELITIS.

By F. KINGSLEY NORRIS, M.D.
Honorary Physician to Children's Department, Alfred Hospital, Melbourne.

THIS afternoon I shall demonstrate the essential treatment of anterior poliomyelitis. As has been indicated to you, convalescent serum therapy, when properly carried out, may be of great value, but whatever other line of treatment we adopt in the early stages, some form of rest is essential.

Dr. Webster's sections have shown how this condition of œdematous hæmorrhagic myelitis may result in changes varying from a temporary dysfunction to utter destruction of the cells of the cord and brain and even the cells of the dorsal root ganglia may be involved. You have also been told that pain and hyperæsthesia may be outstanding symptoms in the early stage of the infection. Both these disturbances, pain and paralysis, call for adequate rest to the whole body.

In the first instance I wish to demonstrate this girl, aged eleven years, who became ill seven days after her brother had died of a rapidly extending form of poliomyelitis. She suffered from headache and neck stiffness, a marked spine sign was demonstrable. Her cerebro-spinal fluid was under slightly increased pressure and revealed 330 mononuclear cells per cubic millimetre, with a normal chloride estimation of 730 milligrammes per hundred cubic centimetres. There was no obvious paralysis or weakness and within twenty-four hours of the onset she received convalescent serum intrathecally and intravenously. She was placed on a firm mattress where she was content to lie quite still; the bed clothes were raised from the legs by a cradle; a pillow was placed under her knees and right-angled splints were applied to her feet. If she had been a smaller child or if we had had any difficulty in keeping her quiet, a plaster bed, as demonstrated to you by Mr. Trumble, would have been applied.

This indicates the minimum of splinting that should be applied to any patient with acute poliomyelitis. Although this girl had no apparent paralysis, we know that had she been allowed to suffer the weight of the bed clothes in the presence of a certain atonia of these patients, some foot deformity would almost certainly have developed. She has remained in this position now for three weeks and so far there has been no demonstrable weakness. I say so far, because we are beginning to realize that the onset of paralysis may be delayed up till four weeks. So she will continue *in statu quo* for at least another week.

From admission we anticipated any weakness and each day her muscle groups are investigated, as has been demonstrated to you. There are some group paralyses, not uncommon, that are often overlooked, the *erector spinæ* group, the abdominal wall group and the lateral rotators of the shoulder and hip.

Any position of the body, a limb or portion of a limb is the result as it were of a guy rope action, a nicely adjusted balance between flexors and extensors, abductors and adductors, medial rotators and lateral rotators, pronators and supinators and, of course, with weakness of one component, the opponent becomes contracted at its expense. The recovery of a weakened or paralysed muscle may be retarded or prevented by this disturbed balance and overstretching. In our paralysed patients our splinting is designed as far as possible to prevent this.

Splinting in many cases can be carried out efficiently, at any rate as a temporary measure, by various forms of plaster moulds, but the double Thomas's hip splint forms the basis of most of our appliances. Years ago a series of these splints was stored in the splint room of each hospital and when needed the one that fitted the patient best, was applied. Now each patient has his own special splint and all of us should be able to send to a splint-maker a drawing from which a properly fitting splint can be made. Here is a Thomas's hip splint. It consists essentially of a stem or, in this case, a double Thomas's two stems, of 3·7 millimetre ($\frac{1}{8}$ inch) gauge steel, a chest wing, a thigh wing and an ankle wing of 2·5 millimetre ($\frac{1}{10}$ inch) gauge steel. The metal is padded with a thin layer of felt and covered with leather. The stems pass downwards just lateral to the posterior iliac spine; the chest wing passes three-quarters round the chest just below the lower angle of the scapula with the arms by the side. The thigh wing comes just below the gluteal fold and the groin, the ankle wing just above the *tendo Achillis*. With the patient lying on the side on a sufficiently large sheet of white paper, with the thighs and legs flexed as required, the outline of the body is traced with a long pencil held vertically. This will represent the stems. The site of attachment of the various wings are marked on the child, and on the stem tracing. Next a strip of malleable lead such as flattened gas piping, is passed around the chest at the position of the wing from nipple to nipple. When the strip has taken the shape, it is slipped down to the level of the umbilicus and rounded off without distortion of the curve; after it is transferred to the paper the form of this wing is traced. The same procedure is carried out with the other two wings. If we decide to add foot pieces these also are traced with a foot wing around the broadest portion of the foot. If a deltoid support is needed, this can be traced with the arm in the desired position and can be extended from the chest wing up the side of the chest to the axilla.

One word about the deltoid paralysis. There is often associated with this group a weakness of the lateral rotators, the *infra-spinatus* and *teres minor*, consequently as well as elevating the arm it should be rotated well outward to overcome the powerful pull of the pectoral adductors and the medial rotating *subscapularis* and *latissimus dorsi* muscles, just as in applying splints to a child with Erb's paralysis. For the back muscles flat rest is essential. There the plaster bed is most useful. The most horrible scoliosis may be produced by a neglected unilateral back paralysis. Here is such a case.

If any deformity is commencing three fifteen centimetres (six inch) canvas strips can be applied, one from the convexity of the curve to one side of the bed and two from the concavity, one well under the arms and one round the pelvis, to the opposite side of the bed. Any abdominal weakness, easily felt as a difference in tone or seen in an attempt to "puff up the tummy," is best checked by a firm binder.[1]

The stage of initial paralysis which is brought about by the œdema causing destruction and temporary dysfunction, may last up till eight to twelve weeks, when this œdema subsides.

The stage of residual paralysis is then reached which is seldom as extensive as the initial stage. Consequently, very often portion of our splinting apparatus can be cautiously discarded by degrees, as muscle reeducation gradually restores the function

[1] A demonstration was then given of the method of measuring the various splints and their application.

of the weakened groups. But hasten slowly. It is difficult to persuade patients or their parents of the necessity for extensive splinting or the slowness in its discarding. Fatigue of the muscles greatly hinders recovery.

This stage takes us up to the end of the second year when some corrective measures may be undertaken, but such procedure is minimized if our earlier treatment has been efficient.

Mr. Kent Hughes will demonstrate the further stage in treatment.

TREATMENT OF ACUTE POLIOMYELITIS.

By W. KENT HUGHES, M.B. (London), M.R.C.S. (England),
Honorary Consulting Surgeon, Children's Hospital, Melbourne.

Rest.

I DO not think the profession is properly seized of the importance of absolute rest in the treatment of acute poliomyelitis. The excellent plaster bed demonstrated to you by Mr. Trumble is a quick and easy method of obtaining partial rest. If you are not skilled in the use of plaster bandages, use it as a background for complete rest, leave a large hiatus in the region of the buttock so that the patient will not have to be lifted out when using a bed pan and when the plaster bed is set, apply plaster bandages to prevent movement of head, limbs and abdomen.

How long should complete rest be insisted upon? Never less than three months in my opinion, and the longer the better in severe cases up to twelve months.

A good deal of confusion exists as to the proper position for the limbs so as to insure as complete rest as possible. T. S. Ellis and others long ago pointed out that midway between the extreme excursion of a limb or portion of it was the position of rest. Thus the proper position for the arm in paralysis of the shoulder muscles is not at a right angle as usually taught, but about 10° short of it. It must be remembered that in no instance are you dealing with a single paralysed muscle, but rather groups of muscles one or some of which are more affected than others. In the case of the knee joint, full extension will pull on the hamstrings and hamper their recovery and so for all joints. It is generally forgotten that the most frequently paralysed muscles are the abdominal, as they are affected both in the upper limb and lower limb cases and nearly always show much want of power as they are generally overlooked, both in the treatment by rest and in after treatment.

Movement, Active and Passive.

Do not be in a hurry to try to see what your patient can do in the way of muscular effort. The earlier you begin such exercises, the more harm will accrue to your patient. Rest after many years will benefit some patients especially in a severe type such as *talipes calcaneus*. Passive movement, such as massage, should not be used in the first three months. One active muscular effort is worth a ton of massage. In bad cases place the patient in a warm bath in order to give the weakened muscle as little to do as possible. In all cases let the movement be such as requires the least exertion. For instance, it is wrong to ask the weakened deltoid to raise the arm through its last 5° of movement. Let the arm be approximated to the chest wall and support it while the patient endeavours to raise it through its first 5° of movement. If it is not possible to place the patient in a warm bath, let him be recumbent and hold the arm from the bed so that no friction has to be overcome. To get the best results means infinite patience and meticulous attention to detail.

Heliotherapy and diathermy are two very useful adjuncts. By heliotherapy I mean complete exposure to sun and air of the whole body back and front. Diathermy is very helpful in severe cases. It is better to apply it to one limb at a time with a short interval between each treatment.

Talipes Equino-Cavus and Talipes Calcaneus.

I shall take two examples of deformity arising from an attack of acute poliomyelitis. That deformity should occur is somewhat of a disgrace and a severe criticism of our method of teaching the art of surgery.

Talipes equino-cavus is perhaps the commonest form of all. It is not due to a shortened *tendo Achillis*, pulling the *os calcaneus* vertically, so much as to the dropping of the foot anterior to the mediotarsal joint. Many cases can be cured without division of the *tendo Achillis* and in the severest type twelve millimetres (half an inch) of lengthening will generally suffice. The sequence of events is that owing to the weakening of the dorsal flexors, the foot is plantar flexed. The plantar flexors contract in the absence of opposition from their opponents and the foot is foreshortened. The fibrous structures of the sole are firmly contracted. The plantar fascia is not merely a thin fibrous band passing from the heel forwards; it is a large compact body occupying the sole which muscles, nerves and blood vessels penetrate. The whole of this structure is contracted and thickened. In order to unfold the foot and overcome the *cavus*, we must divide it thoroughly in two planes. First, strip the superficial layer from the structures deeper to it right across the foot from the inner to the outer border as far forwards as the superficial part has any connexion with the muscular septa. In severe cases it will be necessary to divide this transversely near the tuberosity of the *os calcis*. In many cases, it will stretch quite easily when thoroughly freed from the bands of fibrous tissue passing to tarsal ligaments.

The next step is to free the attachments of the deep portions of the plantar fascia to the tarsal ligaments. This is done by stripping the *abductor hallucis* from the first metatarsal bone and cutting off its attachment posteriorly to the superficial plantar fascia. By means of broad periosteal elevators proceed across the sole keeping close to the bones.

Divide all the bands of fascia you encounter and finally divide the long and short plantar ligaments. The *cavus* can generally be unfolded at this stage and the anterior portion of the foot restored to its proper relation to the posterior. If full dorsal flexion cannot be obtained, it is due to three main causes: (i) shortening of the *tendo Achillis*; lengthen it by a Σ-shaped incision; (ii) resistance of the skin on the medial surface of the posterior portion of the foot; (iii) contraction of the capsule of the talo-navicular joint; open the capsule freely on the plantar aspect.

Talipes Calcaneus.

Talipes calcaneus presents a more difficult problem in that we have to deal with more severe and widespread involvement of muscles. In slight cases it is sufficient to attempt to rectify the deformity by means of plaster bandages. In young children the bandage is placed above the knee (the limb is bent to a right angle), passes down the outer side of the limb round the *calcaneus* and over the knee on the inner side. A strong pull is made to try and replace the *calcaneus*. Three times is the manœuvre repeated and then the action is reversed. Begin from the inner side of the thigh, down the inner side of the leg to the *calcaneus* and proceed up the outer side to above the knee. When the plaster is almost set, apply a bandage to the anterior portion of the foot to reduce the *cavus* which always accompanies *talipes calcaneus* and which is of a sharper curve than that met with in conjunction with *talipes equinus*.

In older patients the bandaging commences below the knee and the plaster is kept in position by a couple of turns at this spot, otherwise the procedure is the same. When this treatment has been carried out for six months and no further improvement is noted, we may then proceed to divide the plantar fascia as in *talipes equinus*. In addition we must free the *calcaneus* from its connexion by free division of ligaments and it will be necessary as a rule to make an incision on the lateral border to perform this thoroughly. When the division of ligament is complete, replace the *os calcaneus* in as near its normal position as possible and put the foot in plaster. If there is a fairly strong peroneal muscle, at a later date I insert this into the plantar surface of the *calcaneus*. In the case of the *peroneus brevis* this can be done by removing a piece of bone at its distal attachment and fixing it in a groove at the anterior part of the plantar surface of the *calcaneus*.

SYMPTOMATOLOGY.

The following extract from a memorandum issued by the conference of medical officers of health of the Melbourne metropolitan municipalities is reproduced in this place in order to make the series of articles more complete. At the course Dr. H. Douglas Stephens gave a demonstration on the same subject.

Mode of Onset.

In most epidemics a small number of cases have occurred in which the onset of paralysis synchronizes with the onset of fever, but fortunately these represent a very small proportion. The majority of patients give a definite history of illness lasting for some days before paralysis developes. Draper's classification of the mode of onset is helpful: (i) Hyperacute cases in which paralysis is not heralded by any sickness; (ii) dromedary type in which after a period of symptoms of general infection, an interval of apparent recovery lasting for two to three days precedes the signs of meningeal involvement which usher in paralysis. This class of history, of an interval of cessation of symptoms between the two "humps" of the disease was often noted in New Zealand and in Melbourne in 1925; (iii) straggling group in which but slight variation occurs in the symptom complex of a sustained and progressive illness.

Stage of General Infection.

A mild degree of fever is the most constant symptom. It lasts throughout the prodromal period in the straggling cases and remits in the interval of the dromedary type. Headache is common and often intense. Constipation has been the rule in recent epidemics, though Krause reported an epidemic in Westphalia in which diarrhœa was a prominent feature. Convulsions are uncommon; repeated convulsions occur very rarely. Vomiting is common at the onset, but seldom persistent. Symptoms of involvement of respiratory and gastro-intestinal tracts may occur, but in recent epidemics complaint of slight sore throat has been more frequent. Examination of the throat reveals engorgement and slight enlargement of the tonsils, but no exudate. Such symptoms may occur with any of the acute infections, yet even at this stage there are certain manifestations which aid in the differentiation from other diseases. Drowsiness and restlessness occurring in the same patient are frequent, the child lying quietly with wilted appearance until approached, when he becomes anxious and apprehensive, resisting any handling with a snarling whine of resentment. This change in disposition on handling contrasts with the apathy of the same patient when undisturbed, an apathy out of all proportion to the temperature. Retention of urine or incontinence and profuse localized or generalized sweating are frequently observed.

Lassitude with mild fever, vomiting and constipation leading on to a state of drowsiness and irritability with headache, sweating and possibly retention or incontinence are enough to justify a tentative diagnosis of poliomyelitis and, in times of epidemic at least, call for careful observation for the first evidence of the stage of meningeal invasion if such is to develope.

Stage of Meningeal Invasion.

Two symptoms are of great diagnostic value at this stage. Pain is felt in the back, in the neck and in the scapular region. In many cases, particularly in older patients, it is severe. Anterior flexion of the spine increases the pain. True head retraction is uncommon, but any attempt to flex the spine anteriorly by bending the head forward or by attempting to flex the hips with the knees extended provokes stiffening of the back and a whimper or cry of protest. For this reason, a restless, tossing child obtains relief when a small pillow is placed under the shoulders to cause a slight degree of opisthotonus. Popliteal pain is common and often persists. Hyperæthesia may be generalized, but usually affects only one or two limbs. It is unaccompanied by swelling. It frequently precedes the onset of paralysis in a limb or muscle group, and may persist for several weeks after paralysis has developed. Tremor of the hands is frequently observed. The reflexes are irregular. Inequality is common, and an increase in the irritative stage may be followed by a decrease in the paralytic. Photophobia is frequently present. Even in the absence of an epidemic the presence of hyperæsthesia, tremor, pain or anterior flexion of the spine, following a general infection as evidenced by fever, headache *et cetera*, justifies the tentative diagnosis of poliomyelitis and calls for proof by examination of the cerebro-spinal fluid. It cannot be too strongly emphasized that promiscuous lumbar puncture

in cases of acute illness is reasonable and unfair, but, in times of epidemic every attack of acute illness for which thorough physical examination cannot reveal the cause should be watched carefully for the first evidence of meningeal invasion and a lumbar puncture done forthwith. For diagnostic purposes care should be taken to remove the smallest quantity of fluid possible. For the cell count two to three cubic centimetres of fluid should suffice. The findings on examination of the cerebro-spinal fluid vary at different stages of the disease, but the pressure is increased and the fluid remains clear or at most "ground glass" in appearance throughout the acute stage. Globulin is increased as is also the number of cells, of which mononuclear cells predominate after the first twelve hours. The count varies from ten to three thousand cells per cubic millimetre and while the increase in cells is valuable in confirming the diagnosis, it cannot be correlated with any prognosis as to the extent of resulting paralysis. At this stage the symptoms may subside as in the true abortive type of case. This also represents the stage beyond which, it seems, the illness does not develope after early serum therapy. Alternatively one of the various types of paralysis may now appear, according to the localization of the pathological process in the central nervous system.

Stage of Paralysis.

The onset of paralysis usually occurs on the second or third day of illness. It is usual to classify the paralysis into divisions as follows:

(*a*) Spinal, affecting the lower motor neurone, the common type,
(*b*) Cerebral—affecting the upper motor neurone, producing a spastic paralysis,
(*c*) Cerebellar, in which ataxia is a predominate symptom,
(*d*) Polyneuritic, in which hyperæsthesia is marked and persists, anæsthesia is uncommon and, if prominent, suggests a diagnosis other than poliomyelitis.
(*e*) Bulbar and
(*f*) Ascending cases in which paralysis spreads upwards, the Landry type.

In most cases the initial paralysis is not exceeded and in all the ultimate residual paralysis tends to be much less than the initial. The recovery of muscle function which results, depends upon the recovery of the circulation to nerve cells as the lymphocytic aggregation and œdema around the vessels subside. The second factor which influences recovery, is the care given to the patient. In those cases in which respiration is interfered with, the drowsiness seen in the early stages gives place to a vivid alertness, all energies being concentrated upon the effort of breathing with any unaffected muscles remaining. The flaccidity and asymmetric distribution of the paralysis are dealt with fully in older literature. Paralysis of the muscles of back, buttock, chest wall and abdomen are easily overlooked. Bulbar types with ocular and facial paralysis are characterized by a profound stupor which may continue for some weeks. The prognosis in this type of case for life and paralysis is good, contrasting with the danger to life in those cases in which the respiratory muscles are involved. Mortality in poliomyelitis is not due to the toxæmia, but to interference with respiration, where the process of cell accumulation is marked around the vessels supplying the respiratory centre.

Reviews.

PROGRESS IN NEUROLOGY AND PSYCHIATRY.

A BOOK that deserves to be far more widely known than it has been in the past is "Nervous and Mental Diseases," by Bassoe, of which the 1927 volume has recently been published.[1] It is in reality a summary of the year's literature in neurology and psychiatry. Its first section deals with nervous diseases and endocrine disturbances and comprises three-quarters of the whole book. Whilst reports of many interesting and rare cases are given, considerable attention is given to the more prominent diseases as *encephalitis lethargica* and its sequelæ, epilepsy, cerebral tumours and cerebral syphilis. The information about these diseases in such small compass is remarkable and extremely useful. Curiously enough the author follows the psycho-analytical school and classifies epilepsy as a neurosis. In all recent editions of this book the beneficial effects of "Tryparsamide" in cerebral syphilis, including general paralysis of the insane is stressed. It seems strange that this drug, although introduced originally in 1918, has so far been ignored in Australia.

The mental diseases section comprises less than one hundred pages and, compared to the neurology section, is poor and parochial. It may be that few discoveries were made in psychiatry during the year; but, nevertheless, we are surprised after reading the excellent concrete summaries in neurology to find the indefinite generalities in psychiatry.

To an Australian the book is disappointing in that no Australian author is quoted and no mention is made of the year's work in sympathetic ramisection.

The merit of this volume, as in previous volumes, lies in its neurology section and to those who seek a mine of information in neurology at a very reasonable price, this work is confidently recommended. Annual volumes form a veritable reference library in nervous diseases and those who have subscribed annually in the past, cherish their volumes and refuse to lend them.

[1] "The Practical Medicine Series Comprising Eight Volumes on the Year's Progress in Medicine and Surgery": Under the General Editorial Charge of Charles L. Mix, A.M., M.D.; Nervous and Mental Diseases; 1927. Chicago: The Year Book Publishers. Crown 8vo., pp. 387, with illustrations.

A PRACTICAL HANDBOOK IN SURGERY.

"REQUISITES AND METHODS IN SURGERY," by Cathcart and Hartley,[1] is a treatise on surgical handicraft more comprehensive than the well-known "Caird and Cathcart" and remarkable for the range of its subjects. The authors describe various methods of examination, analysis of gastric contents, barium meal technique, cystoscopy and many others. Chapters are devoted to case records, anæsthesia, preparation and after-treatment for operations, bandaging, wounds, shock, sprain, emergencies, medico-legal notes and so on. The section on sterilization includes a discussion of the various types of steam sterilizers. Hints are given on preparing a room in a private dwelling for use as an operating theatre and the surgeon is given details and a plan for the building and equipment of his own theatre from the simplest materials. Directions are given for tying knots, making autopsies, microscopical sections and preparing pathological specimens. Hints are given about artificial limbs and trusses. The chapters on fractures and joint tuberculosis well illustrate the esentially practical nature of the book; they include the methods of measuring and the directions which should be given to the village blacksmith for the preparation of splints. Suggestions are given for the occasions on which a medical practitioner may be forced to provide makeshifts for more desirable equipment. Thus powdered ice and salt are suggested as a means of local anæsthesia, a kitchen chair for maintaining Fowler's position and saw-dust and charcoal are prepared as surgical dressings. A special chapter is devoted to hospital economy.

The treatment of acute infections within the abdomen is described in one line: "Operate at once to remove the source of infection," advice which would be questioned by many surgeons with regard to the Fallopian tubes or gall bladder.

The book is a source of very useful information much of which is not readily available in the textbooks, and should be welcomed alike by the student and house surgeon, the general practitioner and the surgeon, but above all it should prove a veritable *vade mecum* for the man in the country who has not the conveniences of a fully-equipped hospital and who cannot easily obtain the assistance of the splint-maker, the masseur and such skilled helpers. A special field of usefulness should therefore await the book in Australia.

[1] "Requisites and Methods in Surgery for the Use of Students, House Surgeons and General Practitioners," by Charles W. Cathcart, C.B.E., M.A., M.B., C.M. (Ed.), F.R.C.S. (England and Edinburgh), and J. N. Jackson Hartley, O.B.E., M.B. (Ed.), F.R.C.S. (England and Edinburgh); 1928. Edinburgh: Oliver and Boyd. Crown 8vo., pp. 484, with illustrations. Price: 12s. 6d. net.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 1, 1928.

Medical Examiners for Life Assurance Societies.

Life assurance societies have sought the assistance of the medical profession in the past in connexion with the greater part of their business. It has been ascertained from actuarial investigation that the risk attaching to insurance of the lives of persons who have been examined by competent medical practitioners and found to be physically sound, is considerably less than that associated with the insurance of persons not subjected to medical examination. When no medical examination is demanded, the proponents are required to sign a declaration to the effect that as far as they are aware they are in good health and of sound constitution. It is obvious that few societies find it expedient to repudiate their liability when it is found that the declaration was false, unless the evidence of deliberate fraud is overwhelming. It is in consequence in the interest of the societies to pay for medical examinations. But as life assurance societies are commercial undertakings, it is unlikely that the payment made to the medical practitioners engaged to carry out the examinations will be a generous one. Many of the larger organizations employ a whole-time referee and find it wise to select a practitioner of experience and resource who is able to command a good remuneration.

The matter of the fee to be charged by general medical practitioners for medical examination and report has been considered from time to time by the several Branches of the British Medical Association in Australia and by the Federal Committee. The view has frequently been expressed that a thorough examination conducted in accordance with modern requirements together with the writing of a full report is worth more than one guinea. It has also been urged that even when a short report suffices, the examination to be of use to the society should be as careful, as thorough and as painstaking as if a full report were demanded. In view of this contention objection has been taken to the reduction of the fee to ten shillings and sixpence. The Branches and the Federal Committee have come to the conclusion that if a relatively high fee were charged, the societies would adopt other measures. Small policies can be accepted without examination; proponents for large insurances can be sent to whole-time referees. In these circumstances it has been determined that one guinea is the recognized fee for life insurance medical examination and report and that a fee of ten shillings and sixpence may be charged if no examination of urine is required and if the report is a short one.

There is a third method of dealing with proponents under special conditions. When the persons live in sparsely populated districts the societies send travelling medical examiners. The usual practice is for the travelling medical examiner to visit a district at a time when the agents have collected a number of proponents. In the past the arrangements were frequently made between the agent and the medical examiner. The Federal Committee finding that the terms and conditions were far from satisfactory, brought the matter to the notice of the Life Offices' Association of Australia and negotiated for certain improvements. At the meeting of the Federal Committee in April of this year it was determined that the financial arrangements should be made between the societies and the travelling medical examiners and not through the agents; that all travelling expenses including railway and steamer fares at first class rates should be paid, that travelling or maintenance allowances be paid including hotel and other expenses and that the remuneration for the actual examination be at the rate of one guinea each with a minimum of ten guineas a week. This should be in addition to the travelling and maintenance allowances. At the recent meeting of the Committee it was further determined that a maintenance allowance of twenty-five shillings a day should be paid. The necessity for a fixed rate of payment becomes apparent when

the conditions obtaining at present are considered. A travelling medical examiner goes into the country, spends a week in one district and examines six proponents. He is paid his railway fare, six guineas for the examinations and ten and sixpence a day for five working days as maintenance allowance. This would amount to eight pounds eighteen shillings and sixpence. He is charged eighteen shillings a day at his hotel, making four pounds ten shillings; he has to pay a further two pounds ten shillings for meals while travelling, for porterage, for gratuities and other incidentals and makes one pound eighteen shillings and sixpence for his week's work. But as he will not be required to travel every week for the society, his income out of which he has to maintain his home, clothe himself and his dependants and pay the premium for his own life insurance will not amount to one hundred pounds two shillings a year. The inadequacy of the remuneration now paid to travelling medical examiners will be brought to the notice of the Life Offices' Association and the claims made on their behalf will be urged. It will be pointed out that even if the remuneration demanded be paid, the medical examiners will not receive a handsome recompense for their labours.

Current Comment.

ACUTE OSTEOMYELITIS.

ACUTE osteomyelitis is one of the conditions occurring in children which always call for prompt and efficient treatment. The extent to which operative treatment should be carried out, depends on the amount of involvement of the bone and this in turn varies according to the stage at which the patient comes under observation. A special discussion on the treatment of acute osteomyelitis was held recently before the Royal Society of Medicine.[1] H. Platt, of Manchester, opened the discussion and confined his remarks to pathology, symptomatology and surgical treatment. He said that the term acute osteomyelitis implies an acute inflammatory lesion affecting all the various anatomical constituents of bone and that the type of acute bone infection under discussion was a metastatic phenomenon, dependent on the existence of a primary bacterial focus elsewhere in the body. The first important point for consideration thus arises, namely the necessity for undertaking a blood culture as soon as the patient comes under observation. Platt does not lay stress on this, though he points out that *Staphylococcus aureus* was responsible for thirty-three out of forty-one infections, the streptococcus for four and *Staphylococcus albus* for one; in three instances the organism was not identified. Alexander Mitchell emphasized the importance of blood culture. Eric Lloyd who analysed twenty-nine cases occurring in the Hospital for Sick Children, Great Ormond Street, gave some particulars of bacteriological examinations in fatal cases. Of eleven children who died, nine yielded a culture of *Staphylococcus aureus* from the wound. The blood of one of these nine yielded a streptococcus, that of another yielded *Staphylococcus albus* and that of two yielded *Staphylococcus aureus*. It is not clear from the table whether blood culture was made in the remaining instances or not. W. H. Ogilvie gave details from the clinical histories of fifty-one patients with acute osteomyelitis, treated at Guy's Hospital. In forty-two of these the causative organism was *Staphylococcus aureus*, in six no culture was made. It is not stated whether blood cultures were made or not. Alan Perry stated that at the London Hospital blood culture yielded a positive result in 75% of cases. The taking of a blood culture is important from several points of view. It is an aid to both diagnosis and prognosis. When the organism obtained on blood culture is different from that recovered at surgical operation, as in several of Lloyd's patients, one or both of the organisms must be regarded as a contamination. It is of interest to note that a staphylococcus has been regarded as the cause of osteomyelitis more frequently than any other organism. Lloyd has not seen a patient with a staphylococcal septicæmia recover.

The second question to be considered is the pathology of the condition. In order that this may be understood it is necessary to study the arrangement of the blood vessels supplying the long bones. It was held by earlier observers that the epiphyseal, metaphyseal and nutrient arteries which supply the cancellous tissues, are end arteries. Platt holds that equal significance must be attached to the extreme tortuosity of the capillaries of the metaphysis which leads to a slowing of the blood current and the arrest of tiny emboli. It will be remembered that a similar explanation was given by Piney of the tendency for malignant metastases to occur in the cancellous tissues of bones. Piney also undertook a series of experiments from the results of which he concluded that the bones are devoid of lymphatics. In view of the fact that osteomyelitis occurs almost exclusively in children and young adolescents, it is important to contrast the blood supply of the growing bone with that of the adult. Platt points out that in the child the dominant vascular region is the metaphysis. As the period of cessation of growth approaches, the vascular picture of the bone changes. The shaft supply from the nutrient artery assumes greater importance and Platt also regards it as probable that free intercommunication developes between systems which were formerly separate. This would doubtless tend

[1] *Proceedings of the Royal Society of Medicine*. June, 1928.

to speed up the circulation and emboli would be less likely to be arrested than in a less actively moving stream. At the same time it is probable that the actively growing bone cells of the young person would be less resistant than those which had reached maturity. The fact that infections occur most frequently in the actively growing metaphysis supports this contention.

Platt refers to the work of the Toronto School, headed by Starr. Starr has shown that while the spread of an infective focus to the surface and so to the superiosteal space, is early and rapid, the cancellous tissue of the metaphysis seems to offer considerable opposition to downward spread towards the medullary cavity. The juxta-epiphyseal focus remains small and localized at a time when considerable stripping of the periosteum has taken place. Platt states that it is not uncommon for the whole shaft to be stripped, while the medullary cavity remains uninvaded. The medullary cavity proper is rarely infected until a much later stage and is then often invaded not directly from the metaphysis, but from the subperiosteal space along the Haversian canals by retrograde infection. Considerations such as these have a distinct bearing on operative treatment. Platt states that it should be the aim of the surgeon to establish early and effective drainage and to achieve early sterilization of the wound. The operation must be so designed that further devascularization of both infected and uninfected bone is avoided and so that infection is not directly introduced into uninfected areas. The systemic infection must be combated by appropriate general and specific treatment. Platt is definite in his condemnation of the "gutter" operation in the earlier stages of the condition. The metaphysis should be opened freely either by multiple drill holes or by removal of a small trephine disc. The "gutter" operation is indicated only when the medullary canal with its lining of cancellous bone is infected widely. Platt has performed a conservative operation on twenty-two patients of the series of forty-one. In fourteen of these healing without sequestration occurred; in the remainder sequestra of various shapes and sizes were extruded spontaneously or were removed at a secondary operation. The operation of diaphysectomy should in his opinion be but rarely performed. Whenever it is used in acute osteomyelitis there must always be a definite risk of failure of regeneration owing to destruction of the periosteum in part or in whole. The literature contains records of many cases in which this has occurred. Ogilvie stated three principles on which he based his operative treatment. The first is that it is impossible at the time of operation to estimate the extent of present or future necrosis in an infected bone. That which is found bare, white and apparently dead, may survive at any rate in part and form the basis of repair. The second principle is that in any operation for osteomyelitis consideration should be paid not only to the immediate problem of infection, but also to that of repair which will follow. The third principle is that drainage only is the immediate necessity.

In this discussion the views expressed were practically unanimous in regard to essentials. The value to the general practitioner of this report is the emphasis laid on the necessity for early diagnosis, the importance of local tenderness, the fact that careful inspection of the long bones in a child whose fever is unexplained, will often reveal a focus of osseous infection, the desirability of taking a blood culture and above all the need for early operation which will not be too drastic, but which will provide adequate drainage.

ENTERITIS AND OTHER INFECTIONS IN INFANTS.

THE report recently published in this journal by Marshall Allan and Bryce of an investigation into the cause of a number of deaths occurring in an obstetric hospital was a good demonstration, if such were needed, of the way in which bacteriological methods of examination may be used to reveal the origin of an infection. Another instructive report has been published by G. F. and G. H. Dick and J. L. Williams of an epidemic in which enteritis was associated with mastoiditis in infants.[1] The epidemic occurred in an institution devoted to the care of homeless infants. Twenty-seven infants died and sixty-one became ill, but recovered. The morbidity rate in the institution was 39·5% and the mortality rate amongst those infected was 30·7%. The illness began suddenly with diarrhœa and loss of weight. Irregular fever was present. The tympanic membrane was incised in eleven instances and the mastoid was drained in five of the patients who died. Surgical drainage of the mastoid did not appear to affect the course of the disease as compared with that in patients not submitted to operation. On bacteriological examination colon bacilli and green-producing streptococci were found in the mastoid and in the blood of several patients. Since both these organisms are normally present in infants, it was concluded that there was not sufficient basis for the assumption that either was the cause of the epidemic. Subsequently Morgan's dysentery bacillus was recovered in five infants who died, after culture on lactose-litmus agar plates was adopted. The children were being fed on powdered milk preparations and these were found to contain living bacilli. Fresh breast milk, cow's milk or protein milk was substituted and was boiled for ten minutes before being given. Bottles were boiled and nurses were required to wear rubber gloves when feeding the infants. The epidemic came to an abrupt end. Apart from the bacteriological lesson to be learned from this report, it must be recognized that nurses who change babies' diapers, should not handle babies' food. Particular care should be taken in this regard in institutions. Gloves are a safeguard, but it is better to have food prepared, as is done in Mareeba Hospital, Adelaide, by a set of nurses different from those who attend to the children's other needs.

[1] *American Journal of Diseases of Children*, June, 1928.

Abstracts from Current Medical Literature.

THERAPEUTICS.

Camphor and Strychnine and the Cardiac Output.

C. P. WILSON, T. R. HARRISON AND C. PILCHER (*Archives of Internal Medicine*, November 15, 1927) have investigated the effects of camphor and strychnine on the cardiac output of intact unnarcotized dogs. The essential function of the heart is to pump blood and before any drug is evaluated in regard to its circulatory action, it is essential to have information of its effects on the cardiac output. Marshall has demonstrated the feasibility of training dogs for cardiac punctures and respiratory manipulations and has emphasized the fact that operations, narcotics and anæsthetics may cause profound alterations in the cardiac output. The authors therefore carried out a series of observations with animals which were not narcotized and which were healthy and "trained." When the struggles of the animals were at all pronounced the determinations were discarded. The consumption of oxygen was determined by the Benedict spirometer and the Bialock mask. Samples of arterial blood and right ventricular venous blood were withdrawn simultaneously and analysed for their oxygen content in the Van Slyke-Neill manometric apparatus. The cardiac output was calculated according to the Fich formula. After one or in many instances two control determinations of cardiac output the drug was administered subcutaneously. It was found that the effects of camphor were inconstant. The cardiac output and consumption of oxygen were not as a rule altered to any significant extent, although decreases and increases sometimes occurred. The effects of strychnine varied according to the amount of the drug administered. Doses of 0·01 to 0·16 milligramme per kilogram of body weight did not cause any change or caused a slight diminution in cardiac output. Intermediate doses—0·02, 0·04 and 0·08 milligrammes per kilogram of body weight—caused an increase in cardiac output. The degree of increase averaged approximately 20%, but varied in different experiments. The consumption of oxygen was increased to a greater degree than the cardiac output. The authors state that the evidence suggests that camphor has no value in the treatment of patients with circulatory disorders and that the value of strychnine in such conditions is improved.

Treatment of Nausea.

R. W. KEETON AND E. S. NELSON (*The Journal of the American Medical Association*, March 3, 1928) believe that the act of vomiting has two distinct stages: (i) that of violent regurgitation of the duodenal contents into the stomach and (ii) that of emesis. They consider nausea to be a sensation due to abnormal duodenal motor activity, resulting in duodenal antiperistalsis and associated with other symptoms due to the straying of the stimuli into the cardio-vascular field. In the treatment of nausea and its related circulatory sensations, the duodenal dysfunction must be reduced quantitatively or changed into peristalsis. This latter result can be secured by the use of cathartics. Either the bromide ion or soluble barbital is effective in inhibiting the reflexes, but the bromide ion is found to be of more clinical value because of its low toxicity and the absence of objectionable circulatory symptoms. A powder consisting of one gramme of sodium bromide, of sodium bicarbonate and disodium phosphate has been found to be of the greatest value in the treatment of the condition.

Calcium Chloride.

L. BLUM AND P. CARLIER (*La Presse Médicale*, February 25, 1928) record some results of treatment of ascites in cirrhosis of the liver with calcium chloride. Three patients were treated and their extreme ascites was relieved by daily doses of twelve to thirty grammes of pure dry calcium chloride continued for three weeks. In two instances an interval of six days was permitted. No salt was allowed during treatment. The urinary excretion rose from one half to three litres or thereabouts after ten days' treatment and the weight decreased by seven or eight kilograms, the abdominal circumference diminished by nineteen centimetres in one instance. Loss of appetite, weakness and constipation were observed in one patient, but these symptoms improved when calcium chloride was discontinued. "Novasurol" and cyanide of mercury in appropriate doses were ineffectual in one patient prior to the administration of calcium chloride. The failure of calcium chloride in some cases of ascites caused by cirrhosis was due to the occurrence of vomiting or diarrhœa, insufficient dosage, renal inadequacy or the lack of salt restriction. Other calcium preparations were less effective than the chloride.

Acetylcholine.

M. VILLARET AND L. JUSTIN-BESAUCON (*La Presse Médicale*, May 12, 1928) record some studies of the effect of acetylcholine in man. The hydrochlorate of acetylcholine is a white powder, soluble in water; intravenous injection lowers blood pressure by dilating arteries and arterioles. On the heart it has a similar effect to that produced by stimulation of the pneumogastric. A stable solution is difficult to obtain; the best consists of ampoules of five and ten centigrammes with anhydrous glucose to stabilize it. Subcutaneous injection of five to fifteen centigrammes is painless and causes only a slight fall of blood pressure; no ill effect is noted. Five patients with Raynaud's disease were treated and they obtained definite relief from pains and circulatory disturbances and healing of trophic effects took place. Five to ten centigrammes were injected daily. The skin of the extremities became pink in half an hour; the effect lasted from twelve to seventy-two hours. Later the injections were reduced to two in the week. In endarteritis (presenile, senile and diabetic) acteylcholine ten centigrammes improved the circulation of the legs when affected with arterial spasm causing coldness or pain. Similar results were obtained in cases of claudication. Three patients with sclerodermia manifested increased warmth and improved circulation after five centigrammes. Trophic ulcers of the legs after amputation in senile subjects were definitely improved. One to two centigrammes of acetylcholine given subcutaneously caused a definite diminution of sweating in tuberculous subjects for a period up to forty-eight hours. In hypertension, cramps, dead fingers, vertigo and head noises were improved and in some senile subjects blood pressure was considerably lowered.

Treatment of Leprosy.

L. ROGERS (*The Practitioner*, April, 1928) discusses recent advances in the treatment of leprosy. Active preparations of chaulmoogra and hydnocarpus oils were tried. Chaulmoogra oil orally was too nauseating; sodium gynocardate (a fatty acid of chaulmoogra oil) injections were followed by disappearance of lepra lesions. Sodium morrhuate prepared from cod liver oil produced definite improvement when injected. Sodium hydnocarpate was then found to inhibit the growth of acid-fast organisms in dilutions of one in one hundred thousand to one in one million, but it caused irritation and thrombosis when given intravenously. More recently non-irritating solutions have been prepared which can be used subcutaneously, intramuscularly and intravenously in 3% or 2% solutions. "Alepol" is one such preparation; 0·5 cubic centimetre increased gradually up to five cubic centimetres and given by injection twice weekly causes no local or general reaction as a rule. If pain and swelling of the skin lesions or pain along the nerves occur, a week's rest is given and the previous dose repeated. Slight reactions should be aimed at and the injections should be continued for six months to two years. Iodides cause severe reactions, which can be relieved by 0·02 gramme of tartar emetic in two cubic centimetres of water given intravenously every other day. Potassium iodide given over periods of some months, has been used with success in the less active stages of the disease and as an adjuvant to injection treatments. By these methods leprosy can be practically cured, if treated early; that is to say all signs of the disease can be removed and signs of recurrence prevented, unless the patient's general health is lowered by other causes. In view of this it is suggested that com-

pulsory isolation should be relaxed in order to encourage patients to report for treatment in the early stages. Careful examination of the household every six months is indicated when a leper is detected, since it is known that 80% of infections are obtained by living in the same house as another leper and in 80% of cases the incubation period is under five years and averages two to three years. By such means leprosy could be practically stamped out among white people in a short time, though in tropical countries the lack of health supervision would make progress slower.

NEUROLOGY.

Familial Form of Encephalitis Periaxialis.

ARMANDO FERRARO (*Journal of Nervous and Mental Disease*, October, November, December, 1927) records three examples of the familial form of *encephalitis periaxialis* fully investigated both clinically and pathologically. The interest of the record lies chiefly in the fact that the three patients were sister and brothers. There were no other children in the family. Such familial incidence of Schilder's encephalitis has not been recorded before. The ages of the subjects ranged between twenty and thirty years, which is unusual, because others think that the malady usually affects children. In brief the clinical features were visual loss, pupillary inequality, pallor of the discs, spasticity of limbs, exaggeration of deep reflexes, tremors, scanning speech, absence of abdominal reflexes and mental disturbance (euphoria, impairment of memory, emotional instability and a paranoid state). The duration of the illness ranged from five to ten years and no cause was discovered. In face of the foregoing clinical outline it is not surprising that all the conditions were diagnosed as disseminated sclerosis. In each instance a histological examination of the brain was made and the distribution of the changes, its essential nature, the appearance of the globoid cells and altered glial cells and other characteristic changes will be found described in detail and discussed. This examination almost establishes the diagnosis. The only other possibility is Pelizaeus-Mehrbacher's *aplasia axialis extracorticalis* which may be a kindred disease.

The Prognosis of Postencephalitic Respiratory Disorders.

W. ALDREN TURNER AND MACDONALD CRITCHLEY (*Journal of Neurology and Psychopathology*, January, 1928) investigated twenty-nine patients in whom almost every variety of known post-encephalitic breathing anomaly was represented. Tachypnœa was commonest, comprising fifteen patients. Of these nine were cured (so far as the respiration was concerned), one died and six remained unchanged. Spells of breath-holding were next in frequency; five patients were affected; of these one was cured, three were improved and one had died. Next came three sufferers from paroxysmal yawning, all unchanged. Then four patients with tics (coughing, blowing *et cetera*) of whom two were improved and two remained as before. One patient alone had bradypnœa. In this instance the breathing became normal but Parkinsonism increased. In no less than twenty of the total patients there happened to be associated Parkinsonism. The existence of Parkinsonism, however, does not unfavourably influence the prognosis as to the respiratory disorder. Numerous physical and psychical stimuli may temporarily influence the severity of the disorder. Improvement, when it occurs, seems to do so spontaneously and independently of treatment.

Symptomatology of Frontal Lobe Lesions.

ERNEST SACHS (*Brain*, October, 1927) bases a communication on a series of twenty-five frontal lobe lesions all verified by operation or autopsy. He first points out that in his opinion the symptoms emphasized by others as of diagnostic value, namely emotional changes, eye ground changes, unilateral tremor of the hand and arm on the side of the lesion, contralateral loss of the abdominal reflex, anosmia and speech disturbances, are in reality unimportant. They were noted in about half his cases only. He goes on to state that there are two other symptoms more frequently present upon which diagnosis can be based. The first is a slight weakness of the lower portion of the face. This was noted in twenty of the twenty-five cases. The second is a mental change, it was noted in twenty-one of the twenty-five cases and outweighs all other symptoms. The change is believed to be so characteristic that it is possible to distinguish it from that arising in any other kind of nervous disorder. Patients are peculiarly indifferent, they seem to have no realization of the seriousness of their condition, and if told they require an operative procedure, do not worry about this at all. At the same time they are not euphoric. They have loss of memory for recent events.

The Diagnosis of Tumours of the Frontal Lobe.

CLOVIS VINCENT (*Revue Neurologique*, June, 1928) writes a long paper on the diagnosis of tumours of the frontal lobe, basing his remarks on observations of fifteen cases all verified by operation or autopsy. He stresses three symptoms, facial paralysis of cerebral type, aphasia of varying degree and a characteristic mental picture. The facial paralysis is slight and has to be looked for. It affects the lower part of the face and may be observed when the lips and naso-labial folds are moved. The naso-labial fold on the affected side may be dropped and set at an obtuse angle. Voluntary opening of the mouth may display asymmetry and so on. As to the aphasia it also is usually not obvious. It may reveal itself merely in a difficulty of finding and saying the names of objects or people. In some instances it is complete, but transitory. Articulation is not slowed, as has been stated by some observers. The mental changes are characteristic and since they appear early, are not attributable to increased intracranial tension. They comprise change of character, dulling of affection and mental activity, disorientation as to place, defect of memory for recent events and mental indifference, the latter showing itself by an apparent inability on the part of patients to appreciate the gravity of their condition. This triad of symptoms being fixed, the diagnosis of frontal tumour is practically certain. Two other symptoms, however, strabismus and ataxia, are regarded as important. The strabismus is commonly due to sixth nerve paralysis. The ataxia is of the kind described by Bruns, manifested in standing and walking and almost always attended by a sense of giddiness. In the matter of differential diagnosis a frontal tumour is often confused with general paralysis of the insane, hysteria, neurasthenia and epilepsy. Softening of the frontal lobe also may be difficult to differentiate, likewise tumours of adjacent parts, that is in the Rolandic region, temporal lobe and hypophysis, also tumours of the cerebellum. There follows an account in detail of the fifteen cases upon which the paper is based and this is profusely illustrated by reproductions of photographs.

Arterial Encephalography.

EGAS MONIZ (*Revue Neurologique*, February, 1928) draws attention to the utility of intracarotid injections of sodium iodide combined with radiography in the localization of cerebral tumours. In the X ray room and under a local anæsthetic the patient receives from five to seven cubic centimetres of a 25% watery solution of iodide of soda, chemically pure, warmed to body temperature and injected *viâ* the internal carotid artery and immediately thereupon a radiograph is made with one-tenth of a second exposure. As a routine and for comparative purposes this procedure is carried out first on one and then on the other side. In doubtful cases patients have been kept on the table during development of the film and a further injection made when necessary, even three and four have been given at one sitting. But when further repetition has been called for, it has been postponed for nineteen days. It is claimed that the injections are harmless; they may, however, induce slight clonic movements of two or three minutes' duration and perhaps more intense on the injected side. Radiographs are reproduced of the right and left hemispheres from one patient. They clearly display the plan of the middle cerebral artery and a blank space on one side which unmistakably localizes a tumour of the temporal lobe subsequently operated upon with success.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Royal Prince Alfred Hospital, Sydney, on June 14, 1928, DR. J. E. V. BARLING, the President, in the chair. The meeting took the form of a series of clinical demonstrations by members of the honorary staff.

Cerebellar Syndrome Following Epidemic Encephalitis.

PROFESSOR W. S. DAWSON showed a patient who was suffering from a cerebellar syndrome following epidemic encephalitis. Early in 1919 this patient at the age of twenty-three years had suffered from severe "influenza." He had been very drowsy and had seen double both during and subsequent to the acute illness. About a year and a half after his illness he had complained of attacks of giddiness when walking, but not while he was at rest. He had also complained of tinnitus. Later on he had become easily fatigued and had complained of a weakness in his legs. He had been admitted to the Royal Prince Alfred Hospital in 1920, complaining of giddiness and a tendency to fall. By this time his speech had become difficult and indistinct and had been characterized by a slurring quality. The physical signs had included a horizontal nystagmus to both sides, a slight intention tremor in his hands and a moderate degree of spasticity in his legs with patellar clonus. The result of the caloric test, when applied to both sides, had been normal.

Three years previously the patient had been in Broughton Hall. He had become deeply depressed after being told that he was incurable and he had made a suicidal attempt. Professor Dawson said that comparison between his state at the time of the meeting and his record at Broughton Hall showed that his physical condition had been stationary for quite three years. Mentally he was bright and almost cheerful and was quite resigned to his disabilities; he cooperated readily in his examination. He was thirty-two years of age. His station was erect and usually steady; he varied from time to time. In regard to vertigo he sometimes felt as though he were in the air when his eyes were shut; at other times things seemed to spin from left to right or to oscillate. He had fallen and had sustained minor injuries. His gait was reeling; he walked on a wide base and was unsteady in turning. On all fours he progressed with a springy exaggerated movement of his legs. Nystagmus was coarse to right and left, but more pronounced towards the right. There was no vertical movement. There was no ocular palsy. Speech was slurring and indistinct. In regard to active movements, slight ataxy was noticed on completion of movement with the right arm in the finger-nose test. Pronation and supination were steady. In alternate pronation and supination the movements were regular and of equal amplitude, but slowly performed. The rebound phenomenon was pronounced in the right arm. Prehension was moderately good with normal grasp and relaxation. The left grip was stronger than the right, but he had been left handed before his illness. He could draw horizontal lines quite accurately, but his execution was slow. The result of the dotting test was moderately good from both elbow and shoulder. Writing was performed slowly and was irregular. In drawing a circle the patient moved his pencil from point to point instead of forming a curve. There was a slight lateral tremor of the head. He could flex his thigh and elevate his leg in a normal manner, but the knee-heel test was performed with ataxy at the completion of the movement; this was more evident in the right leg.

Dr. C. A. Verge had been kind enough to carry out the vestibular tests. In regard to rotation horizontally there was nystagmus to the opposite side with a very slight degree of past pointing; vertically there was normal nystagmus and past pointing. The result of the caloric test was normal on both sides. No reaction had been obtained to the Wassermann test in both blood and cerebrospinal fluid. Professor Dawson said that he was indebted to Dr. Evan Jones for bringing to his notice the patient who presented quite an uncommon sequel of encephalitis.

Marble Bones.

DR. PHILLIP PARKINSON showed some skiagrams illustrating the condition known as marble bones. He said that it had also been called osteopetrosis and other names. He thought that probably the most apt term would be porcelain bones, since they were very fragile and at the same time very hard. The surgeon who had plated the femur of the patient, had told Dr. Parkinson that he had not previously had so much difficulty in making drill holes in any bone. Dr. Parkinson drew attention to the fact that every bone shown, throughout the whole skeleton, contained the very densest deposit of calcium which almost entirely obliterated all the cancellous tissue. The condition was very rare; so far as he knew the total number of cases reported was less than twenty. The condition was evidently due to a fault in the mechanism controlling calcium deposits in the body, probably the parathyreoid glands. The interesting point about the patient's history was that for many years he had been apparently quite well and had been able to enlist and to do valuable service. The evidence of his service was seen in the skiagram of the thorax, for a large foreign body was present. When the patient had returned from active service in 1918, he had had a gunshot wound of the left thigh which practically never healed, and many sequestra had been removed at various times. He had then fallen over and fractured his thigh; no union had occurred and at last the bone had been plated. There was also a pathological fracture of the neck of his femur.

Acanthosis Nigricans.

DR. E. H. MOLESWORTH showed a patient who was suffering from *acanthosis nigricans*. An account of this case will be published in a subsequent issue.

Retroperitoneal Myxofibroma.

ACTING PROFESSOR B. T. EDYE showed a married woman, aged thirty-five years. For eighteen months she had noticed that her abdomen was enlarging, until it corresponded in size with that of a seven months' pregnancy. She had suffered no pain and no menstrual or urinary disturbances had been present. She had always suffered from constipation and had had occasional attacks of vomiting during the previous ten years. On examination the patient had looked well, although she was thin. The abdomen had been prominent more on the right side than on the left. On palpation a large uniformly firm tumour had been outlined. It had extended from the pelvis to a point midway between the umbilicus and xiphisternum. On vaginal examination the cervix had been normal and the fundus had appeared to be retroverted and displaced to the left. In the right fornix there had been found a firm mass which was continuous with the abdominal tumour. At operation it had been necessary to extend the subumbilical incision upwards in order to gain entrance to the peritoneal cavity. This had been due to the high level to which the retroperitoneal reflexion was raised by the tumour which had proved to be retroperitoneal in origin. The pelvic organs had been normal except for their displacement by the tumour. When separated from its surroundings the tumour had been found to have an attachment of about 3·75 centimetres (one and a half inches) in diameter in the region of the upper and anterior portion of the right *obturator foramen*. There had been considerable loss of blood during the operation, but the patient had made a satisfactory recovery and when last seen had been in perfect health.

The tumour had been examined by Dr. A. H. Tebbutt. Its dimensions were 25 × 23 × 9 centimetres and it weighed 3·46 kilograms (seven pounds twelve ounces). It was encapsuled and there were large vessels in the capsule. The cut surface was firm, greyish-yellow, more or less homogeneous, but contained small irregular spaces filled by gellatinous material. In some of the peripheral parts lamination was present. Microscopically the structure was that of a myxofibroma in which the intercellular material was very abundant and in which the cells were both those of a myxoma and of young fibrous tissue. The matrix in some areas was hyaline, but more often there was a network of fine fibrils with clear spaces between. The vessels were mostly thin walled, just an endothelial lining, though some vessels with thicker walls were seen.

Myeloid Sarcoma of the Fibula.

Acting Professor Edye also showed a male patient, aged twenty years, a plumber by occupation. He had noticed a swelling for six weeks on the lateral aspect of his right leg just below the knee. At first this had been small, firm and painless, but it had gradually increased in size until it reached that of a tennis ball, the enlargement being accompanied by a dull aching pain which, however, was never sufficient to worry him. On examination there had been a rounded swelling in the position of the upper end of the right fibula. It had been firm and painless. The temperature and pulse had been normal and the general health satisfactory. The skiagraphic appearances had been those of a myeloid sarcoma of the upper end of the fibula. At operation the upper extremity of the fibula had been resected. The shaft of the bone had been divided below the tumour with a Gigli saw and removal had been completed by division of the ligaments of the proximal tibio-fibular articulation. There had been some difficulty in preserving the common peroneal nerve which was stretched over, and in places partly imbedded in, the tumour. The wound had healed by first intention and recovery had been followed by no obvious impairment of function.

The tumour had been examined by Dr. A. H. Tebbutt. It occupied the head of the fibula and measured $6 \times 6 \times 5$ centimetres. There was no osseous tissue in the tumour, except a thin layer beneath the periosteum. The superior articular surface was intact, but consisted of cartilage only. The interior of the growth, though uniform, was not much firmer than inspissated caseous material and could easily be scraped away. Microscopically there were numerous multinucleated giant cells in a stroma which was very cellular. The structure was that of myeloid sarcoma (benign giant cell tumour of bone).

Cerebral Aneurysm.

DR. E. W. FAIRFAX reported in detail the history of a patient who suffered from cerebral aneurysm, and described the *post mortem* findings. This report will be published in a subsequent issue.

Hæmorrhagic Septicæmia in a Monkey.

DR. A. H. TEBBUTT gave some interesting details of hæmorrhagic septicæmia occurring in a monkey. The work on which his remarks were based had been carried out during his investigation into the Bundaberg fatalities.

Aneurysm of the Aorta.

DR. PHILLIP PARKINSON, on behalf of Dr. W. B. Dight, showed skiagrams taken from a patient who was suffering from aneurysm of the aorta. It was pointed out that the diagnosis was placed beyond doubt by the stereoscopic skiagrams which were shown. The use of a single skiagram sometimes left some uncertainty as to the condition which was present. X ray examination on May 10, 1928, had revealed the presence of a shadow to the right of the junction of the ascending and transverse portions of the aortic arch in which no pulsation could be detected. The appearance had suggested a primary new growth or a hydatid cyst of the lung, although the latter was less likely. It was thought that the possibility of an aneurysm containing blood clot could not be excluded. When stereoscopic skiagrams had been taken on June 14, 1928, the nature of the condition had been made clear.

Chronic Myocarditis with Gallop Rhythm.

DR. MARK LIDWILL discussed the history of a patient who suffered from chronic myocarditis with gallop rhythm, and explained the tracings which had been obtained by the electrocardiograph. This report will be published in full in a subsequent issue.

Disseminated Sclerosis.

DR. ALLAN S. WALKER showed a patient who was suffering from disseminated sclerosis. He said that the history of the patient was interesting in that it illustrated how a common diagnostic error in this disease could be made with the eyes open.

The patient, a healthy looking school girl of sixteen years, of more than average intelligence, had first come under observation with a history of irregularly periodic "fits." These attacks had occurred only at school and had been described at second-hand by her mother. The girl had become apparently unconscious and latterly had displayed stiffness, twitching and convulsions of the limbs. She had never hurt herself nor bitten her tongue nor passed urine in these attacks, which were heralded by ringing in the ears and the sound of bells. Eighteen months prior to this she had fallen about twenty feet and had sustained slight concussion.

The girl had made no complaint save that she tired readily. She was rather emotional. Careful examination had revealed no neurological or other anomaly save that in his notes Dr. Walker had recorded "slight blurring of the left optic disc."

She had been admitted to the Royal Prince Alfred Hospital with the diagnostic suggestions of epilepsy, cerebral tumour or disseminated sclerosis. The blood serum had not responded to the Wassermann test, skiagrams of the skull had revealed no abnormality and repeated careful neurological examination had disclosed no variation from normal. Neither swelling nor blurring of the outlines of the optic discs had been present. While in hospital, she had had several attacks and had been seen in them by experienced members of the resident medical and nursing staffs. These "fits" had appeared to be of definitely hysterical type. She had also produced blood spitting by scratching the pharyngeal wall and skin lesions by abrading her arms. The fact that her home conditions were far from tranquil had supported Dr. Walker's rather hesitant diagnosis of hystero-epilepsy. He had given a good prognosis and had strongly suggested complete recovery to her. She had remained well for eight months, but had then returned complaining of eye-strain at school. To Dr. Thomas Butler Dr. Walker owed a very complete report on her eyes. Dr. Butler had found one diopter of hypermetropia refractive error, but only $^{6}/_{18}$ vision, definite blurring of both discs, 40° contraction of the temporal side of the visual field of the left eye, though no central scotoma, and he had suggested disseminated sclerosis as the cause. Reexamination had revealed slight but definite pyramidal tract involvement; the right leg and left arm had shown some loss of power and early spasticity, the abdominal reflexes had been absent, there had been bladder precipitancy and she had complained of occasional diplopia.

Pyrexial (vaccine) therapy and intravenous injections of calcium chloride and "Novarsenobenzol" had seemed to effect some definite improvement or at least to hasten a remission, but the patient complained of some pain in the limbs and the gait was a little stiff and dragging.

On reviewing the history he found that at the first interview, though she gave no history of either giddiness or double vision, in response to leading questions she had said that she had experienced both symptoms. Perhaps had the visual fields been tested by perimetry originally, instead by the usual rough clinical method, a positive sign of eye trouble might have then been found. Probably optic disc swelling had been present on the date of the first examination, but was then transitory.

This case showed the remitting nature of the disease, and emphasized the importance of the early affective symptoms. It exemplified well the early diagnostic triad recently stressed by Kinnier Wilson—emotional over-action, euphoria and eutonia. Could this combination be better described than by this patient's mother? The quotation was practically *verbatim* and was jotted down by Dr. Walker just before the girl's second admission to hospital: "I can't make her out. She laughs and cries at nothing. She looks well and she says she doesn't feel ill, but she can't do anything without getting knocked up." It was worth noting that Charcot's triad of symptoms, tremor, nystagmus and scanning speech, was absent in this patient.

Glossitis.

DR. ERIC SUSMAN showed three patients who were suffering from glossitis of nine months' duration. In all three instances the condition was monosymptomatic, in other words the patients had come under observation for sore tongue alone. The first patient suffered from Addisonian anæmia with a characteristic atrophic Hunterian glossitis. It was noteworthy that the patient was able to carry out

all her domestic duties in spite of the fact that her erythrocyte count was just two millions. The second patient had a tongue very similar to that of the first patient; it was reddened, smooth and shiny. The blood count was normal. A strong reaction had been obtained to the Wassermann test. The third patient had a tongue with deep fissures which tended to be smooth and glossy at the periphery. The blood count was normal and a strong reaction had been obtained to the Wassermann test. All three patients had achlorhydria. The second and third patients had had intensive antisyphilitic treatment. Symptomatically they were cured, but from a serological point of view treatment had been a failure. Dr. Susman asked for an opinion as to the specificity of the condition in the first two patients. He wanted to know whether the lesions were syphilitic or whether the patients were merely candidates for Addisonian anæmia.

A Non-motile Strain of Bacillus Typhosus.

DR. A. B. LILLEY described an unusual infection with a non-motile strain of *Bacillus typhosus*.

Osteoporosis of the Hand.

DR. R. ANGEL MONEY described the findings in a case of osteoporosis of the hand following injury. This report will be published in a subsequent issue.

Skiagrams.

DR. H. R. SEAR showed a series of skiagrams taken from patients suffering from pathological conditions of the gall bladder.

Books Received.

FEVER, HEAT REGULATION, CLIMATE AND THE THYROID-ADRENAL APPARATUS, by W. Cramer, Ph.D., D.Sc., M.R.C.S.; 1928. London: Longmans, Green and Company, Limited. Royal 8vo., pp. 163, with illustrations. Price: 15s. net.

FASCIAL GRAFTING IN PRINCIPLE AND PRACTICE: AN ILLUSTRATED MANUAL OF PROCEDURE AND TECHNIQUE, by H. C. Orrin, O.B.E., F.R.C.S. (Edinburgh); 1928. Edinburgh: Oliver and Boyd. Royal 8vo., pp. 92. Price: 7s. 6d. net.

THE BLOOD PLASMA IN HEALTH AND DISEASE, by J. W. Pickering, D.Sc. (London); 1928. London: William Heinemann (Medical Books Limited). Demy 8vo., pp. 258. Price: 12s. 6d. net.

ON THE DYSENTERIES OF INDIA, WITH A CHAPTER ON SECONDARY STREPTOCOCCAL INFECTIONS AND SPRUE, by Hugh W. Acton, Lieut.-Col., I.M.S., and R. Knowles, Lieut.-Col., I.M.S.; 1928. Calcutta: Thacker, Spink and Company. Crown 4to., pp. 192, with illustrations. Price: Rs. 7.

Diary for the Month.

SEPT. 4.—Tasmanian Branch, B.M.A.: Council.
SEPT. 5.—Victorian Branch, B.M.A.: Branch.
SEPT. 5.—Western Australian Branch, B.M.A.: Council.
SEPT. 5.—South Sydney Medical Association, New South Wales.
SEPT. 6.—South Australian Branch, B.M.A.: Council.
SEPT. 7.—Queensland Branch, B.M.A.: Branch.
SEPT. 11.—Tasmanian Branch, B.M.A.: Branch.
SEPT. 11.—New South Wales Branch, B.M.A.: Ethics Committee.
SEPT. 12.—New South Wales Branch, B.M.A.: Nomination of Candidates for Federal Committee.
SEPT. 13.—Victorian Branch, B.M.A.: Council.
SEPT. 13.—New South Wales Branch, B.M.A.: Clinical Meeting.
SEPT. 14.—Queensland Branch, B.M.A.: Council.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii.

ADELAIDE CHILDREN'S HOSPITAL: Medical Superintendent.
COMMONWEALTH OF AUSTRALIA: Medical Officer.
KYNUNA DISTRICT HOSPITAL: Medical Officer.
RENWICK HOSPITAL FOR INFANTS, SUMMER HILL: Resident Medical Officer.
ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, CAMPERDOWN, SYDNEY: Registrar and Senior Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. Marrickville United Friendly Societies' Dispensary. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Contract Practice Appointments in South Australia. Booleroo Centre Medical Club.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to position at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor." THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.